VOLUME I—NUMBER 2 FEBRUARY 1950

UNITED STATES ARMED FORCES

MEDICAL JOURNAL

Published monthly by the Department of Defense

JOSEPH L. SCHWARTZ, *Captain (MC), U.S.N.*, Editor in Chief
WAYNE G. BRANDSTADT, *Lieutenant Colonel MC, U.S.A.*, Associate Editor
ALFRED A. GENTILCORE, *U.S.A.F. (MSC)*, Associate Editor

THE PRINTING OF THIS PUBLICATION HAS BEEN APPROVED BY THE DIRECTOR OF THE BUREAU OF THE BUDGET, OCTOBER 19, 1949.

Streptomycin in Pulmonary Tuberculosis

Treatment of Recent Posthemoptoic Spreads

NORMA C. FUERTOS, *Lieutenant Commander (MC) W. U. S. N. R.*[1]

DURING our studies of the effect of streptomycin upon various types of pulmonary lesions, seven patients were selected for treatment because of recent posthemoptoic spreads. Each of the seven, by chance, was given a different amount of streptomycin. Each, as a consequence of hemoptysis, showed evidence of progressive tuberculosis. Prior to treatment, their clinical condition was critical, or nearly so, and the prognosis extremely poor. As a result of streptomycin therapy four patients showed dramatic improvement, one moderate improvement, and two were complete failures.

Hemoptysis is commonly followed by a bronchogenic dissemination of blood containing tubercle bacilli into a new portion of lung, resulting in a local pneumonitis which is made up of two components; a hemoptoic component due to the blood per se, and an exudative tuberculous component. A high fever and evidence of acute toxicity occurring within a day or so of the hemoptysis is indicative of the hemoptoic component; this may largely resolve within a comparatively short

[1] From the Streptomycin Study Unit, U. S. Naval Hospital, Corona, Calif.

time. A persistence or steady increase in the new area of infiltration after the acute episode has subsided indicates a persistence or progression of the tuberculous seeding. This may be indicated clinically by fever and continued weight loss. Although the bleeding had stopped in each of our seven patients they showed evidence of progression of the posthemoptoic lesion.

CASE REPORTS

Case 1.—A 49-year-old Filipino man was admitted on 10 July 1946, with an old fibrocaseous lesion in the right upper lobe. A right pneumothorax was induced but proved ineffective. During December 1946 the reexpanding pneumothorax was complicated by a tuberculous empyema followed by a bronchogenic spread to the left midlung. The empyema subsided but the bronchogenic spread slowly progressed. On 2 May 1947 there was a moderate hemoptysis and fever, which soon reached 103° F. A chest roentgenogram revealed a posthemoptoic spread to the left base. He received 30,000 units of penicillin every 3 hours from 8 May to 17 May, during which time the acute symptoms subsided. In spite of this, the dyspnea progressed, he lost weight rapidly, and his condition became critical. Serial chest roentgenograms showed increase in the extent of the posthemoptoic spread. On 20 May 1947, 18 days after the hemoptysis, streptomycin therapy was begun (fig. 1). He received a total of 2 gm. daily in 5 intramuscular injections. This was continued for 120 days, ending 24 September 1947. The course was marked by only fair symptomatic improvement because of severe symptoms of vestibular nerve damage; these symptoms persisted for some time after streptomycin had been discontinued. There was apparent complete loss of the vestibular nerve function after the fifty-seventh day of therapy. The sputum remained positive during the period of treatment. The tubercle bacilli continued sensitive to streptomycin until some time after the fifty-sixth day of treatment, and were highly resistant shortly before treatment was terminated. There was distinct resolution of the posthemoptoic spread in the left lung as well as of the preexisting bronchogenic spread (fig. 2).

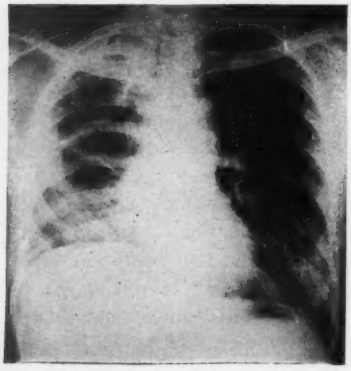


Figure 1.—Case 1. 20 May 1947. The posthemoptoic spread occupies the lower portion of the left lung.

Figure 2.—Case 1. 25 September 1947. After 120 days of streptomycin (2 gm. daily) the spread shows distinct resolution.

This patient has been observed for 26 months during which time the improvement has been gratifying. The last positive sputum was on 15 October 1947; since then repeated monthly sputum and gastric cultures have been negative. The remaining infiltration in the left base continued to resolve and the left lung has been clear since January 1948. The patient compensated earlier for the loss of vestibular function and is slowly regaining the response to caloric stimulation. He is ready for discharge.

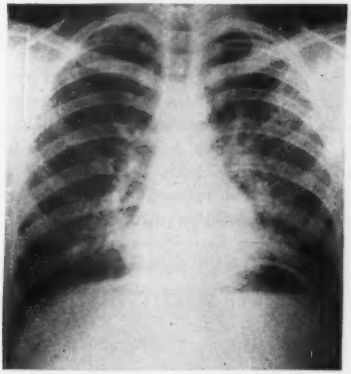


Figure 3.—Case 2. 19 August 1947. The posthemoptoic spread occupies the lower two-thirds of left lung and right midlung.

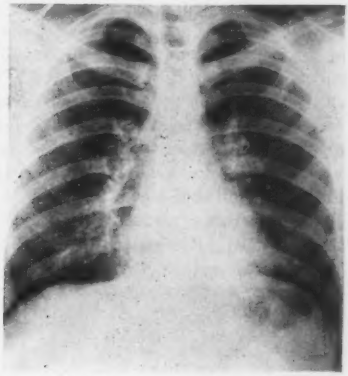


Figure 4.—Case 2. 30 December 1947. After 120 days of streptomycin (1 gm. daily), the spread has completely cleared.

Case 2.—An 18-year-old white man was admitted on 12 June 1947 because of sudden hemoptysis. The next day his temperature was 103.5° F. and he had chills and sweats. A chest roentgenogram on 14 June showed a soft lesion with a small cavity in the left second anterior interspace, scattered exudative infiltration of posthemoptoic origin throughout all the left lung, and extensive bronchogenic spread to the right midlung. He continued to hemorrhage intermittently until 28 June. He received penicillin from 14 June to 3 July during which time the acute symptoms subsided. After this he continued to have a low-grade fever and by 24 July he had lost 24 pounds. On 31 July, crushing of the left phrenic nerve was done. Serial chest roentgenograms then showed progression of the bilateral posthemoptoic infiltration.

On 3 September 1947, 2½ months after the onset of the hemoptysis, streptomycin therapy was begun (fig. 3). He received 1 gm. streptomycin daily, in 2 divided doses 12 hours apart for 120 days, ending 1 January 1948. By the end of this period there was symptomatic improvement, a weight gain of 20 pounds, complete resolution of the extensive posthemoptoic spread, and closure of the cavity. The only remaining roentgenographic finding was a residual fibrotic lesion in the left second anterior interspace (fig. 4). The only manifestation of drug toxicity was a temporary decrease in the caloric response. The sputum and gastric cultures were converted early during the period of treatment, and the only subsequent positive finding was a single gastric culture obtained 1 month after therapy was discontinued. The tubercle bacilli were still sensitive to streptomycin. The patient continued to improve. He was finally transferred to a Veterans' Administration hospital 27 February 1948.

Case 3.—A 42-year-old Filipino man was admitted 4 October 1946. A chest roentgenogram showed a dense shadow in the left midlung suggestive of local atelectasis. A 2-cm. cavity was present in this lesion. Bronchoscopic examination showed extensive ulceration and almost complete stenosis of the visible portion of the left main bronchus. He received 2 gm. streptomycin daily for 120 days from 11 June to 2 October 1947, with complete clearing of the endobronchial lesion, evidence of considerable roentgenographic improvement but with indifferent symptomatic response. High bacterial resistance as well as complete loss of vestibular function was present before the end of therapy.

On 6 December 1947, 2 months later, there was frank hemoptysis followed by an acute febrile course. A chest roentgenogram on 11 December (fig. 5) revealed a massive bilateral hemoptoic spread; by this time the patient was critically ill. On 13 December, 1 week after the initial hemoptysis, streptomycin retreatment was begun. He received 1 gm. daily in 2 divided injections, 12 hours apart. There was no symptomatic response. The patient's downhill course continued. There was first some clearing of the recent spread, due perhaps to resolution of aspirated blood, followed by progression of the tuberculous component. Streptomycin was discontinued 3 February 1948 after 53 days of ineffective treatment (fig. 6). The patient died 1 March 1948.

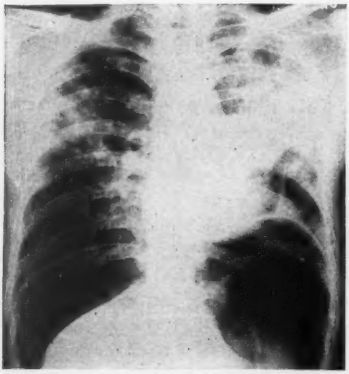


Figure 5.—Case 3. 11 December 1947. An extensive posthemoptoic spread is present in both upper lobes. High bacterial resistance.

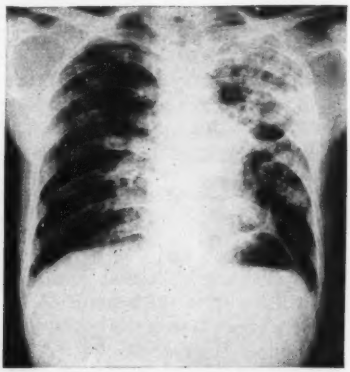


Figure 6.—Case 3. 3 February 1948. After 53 days of retreatment (1 gm. daily), there was persistence and increase of the tuberculous component.

This case demonstrates the apparent futility of streptomycin in the presence of high bacterial resistance.

Case 4.—A 37-year-old Filipino man was admitted to the sick list aboard ship on 26 September 1946, because a routine chest roentgenogram revealed a fibrocaseous lesion containing a small cavity in the right upper lobe. Right pneumothorax having failed, crushing of the right phrenic nerve was done and pneumoperitoneum was induced on 27 November 1946. He was apparently doing well until 1 October 1947 when hemoptysis followed by fever and an extensive posthemoptoic spread to the right base and to all of the left lung occurred. He did not become acutely ill at the time, but there was progressive dyspnea. By

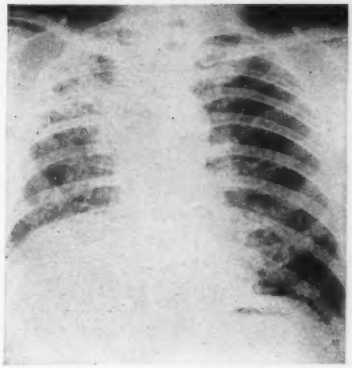


Figure 7.—Case 4. 5 December 1947. The posthemoptoic spread is in the right base and throughout the left lung.

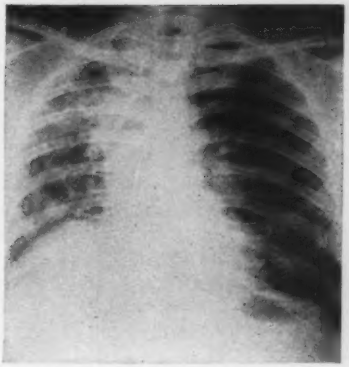


Figure 8.—Case 4. 1 April 1948. After 120 days of streptomycin (one-half gm. daily), the spread has almost completely cleared.

5 December 1947, 2 months after the initial hemoptysis, when streptomycin therapy was begun, he was critically ill. Serial chest roentgenograms showed an increase in the bilateral posthemoptoic infiltration (fig. 7). He received the daily dose of one-half gm. stroptomycin given in single injections for 120 days, ending 3 April 1948. By the end of the 120-day period he no longer appeared ill, and had regained his normal weight. There were no symptoms of drug toxicity. The posthemoptoic spread had almost entirely cleared (fig. 8). Although the sputum remained intermittently positive, bacterial resistance to streptomycin did not develop.

He has been observed for 20 months, during which time a 3-stage right thoracoplasty has been done. There has been no relapse. Sputum and gastric cultures have been negative for the past 6 months.

Case 5.—A 34-year-old white man was admitted on 14 July 1947, with exudative infiltration and early cavitation in the right first and second anterior interspaces. There was also a minimal bronchogenic spread to the left midlung. Right pneumothorax was ineffective, so this procedure was abandoned in favor of pneumoperitoneum which was induced 5 April 1948. Meanwhile the bronchogenic spread in the left midlung was increasing in extent. Hemoptysis occurred 23 April 1948, followed in 2 days by high fever. By 29 April a posthemoptoic spread was evident in the left lower lobe. Penicillin was administered from 4 May to 11 May during which time the acute symptoms subsided. In spite of this the general condition of the patient grew steadily worse. Serial chest roentgenograms showed progression of the new spread. On 25 May 1948, 1 month after the initial hemoptysis, streptomycin therapy was begun (fig. 9). He received 1 gm. daily in single injections for 42 days, ending 5 July 1948. By the end of this 42-day period the clinical and roentgenographic improvement was no more than fair. There were no symptoms of drug toxicity. The sputum remained positive for tubercule bacilli and these continued to be sensitive to streptomycin. After this the improvement continued until by the one hundred and twentieth day after streptomycin was begun the posthemoptoic spread had entirely cleared, and in addition there was evidence of resolution of the previously

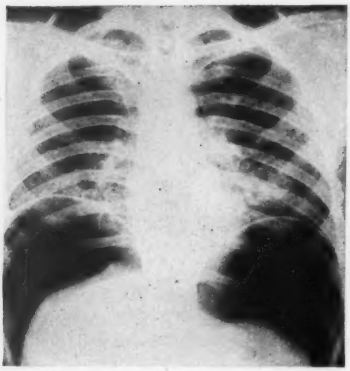


Figure 9.—Case 5. 25 May 1948. The posthemoptoic spread is in the left lower lobe.

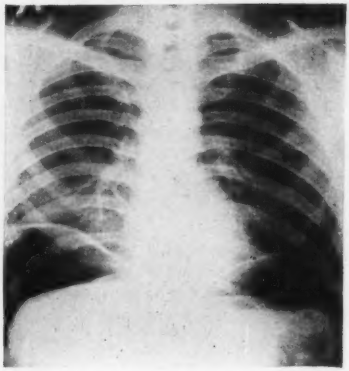


Figure 10.—Case 5. 21 September 1948. After 120 days (1 gm. streptomycin daily for the first 42 days), there is clearing of the spread.

progressive infiltration in the left midlung (fig. 10). In fact, this patient did as well as those given streptomycin for the entire 120 days.

He has been observed for 14 months, during which time the sputum and gastric cultures have been negative.

Case 6.—A 57 -year-old white man was admitted 25 April 1948, complaining of progressive fatigue, weight loss, and shortness of breath during the past year. The chest roentgenogram showed fibrocaseous infiltration with cavitation in both upper lobes. The hospital course was not unusual until 13 May when hemoptysis, with repeated hemoptoic episodes during the next few days, occurred. On

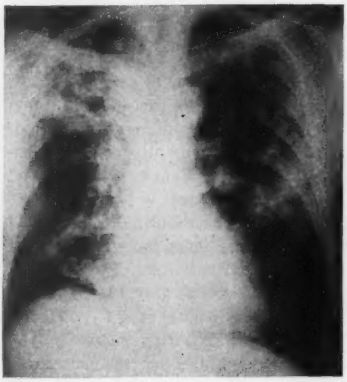


Figure 11.—Case 6. 1 June 1948. A posthemoptic spread occupies the left midlung and right base.

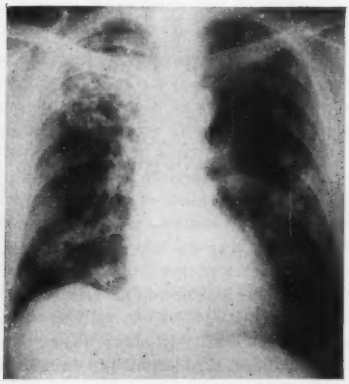


Figure 12.—Case 6. 28 September 1948. After 120 days (one-half gm. streptomycin daily for the first 42 days), there is moderate clearing of the spread.

20 May, his temperature was 101.8° F., and following this the fever continued and the patient rapidly lost weight and strength. Serial chest roentgenograms revealed a posthemoptoic spread to the right base and left midlung. On 1 June, 12 days after the onset of the acute course, streptomycin was administered (fig. 11). He received one-half gm. daily in single injections for 42 days, ending 12 July. By the end of the 42-day period there was considerable symptomatic improvement together with moderate resolution of the bilateral posthemoptoic spread. The sputum remained positive and the tubercle bacilli continued to show sensitivity to streptomycin. Following the forty-second day there was further resolution of the residual spread but by 120 days after streptomycin was first begun, cavitation was evident in the left midlung lesion (fig. 12).

Considering it quite possible that this patient had received insufficient streptomycin, a second 42-day course was given beginning 21 September 1948, using the same dose of one-half gm. but with no further improvement. Again bacterial resistance did not develop. The patient has been observed for 13 months and still shows no improvement.

DISCUSSION

In comparing the symptomatic response under the various dosages employed, most noteworthy was the very slow improvement in the patient who received the daily dose of 2 gm. of streptomycin; this was undoubtedly due to toxicity of the drug. The 2-gm. dose is more toxic to some patients than to others. The symptoms of vestibular nerve disturbance may be particularly troublesome; interfering with appetite, weight gain, and general physical rehabilitation. The one-half gm. daily dose was hardly toxic, while the 1-gm. dose was only slightly more so.

With respect to the roentgenographic improvement seen in those patients which responded to streptomycin, it was evident that all the regimens were equally effective, with exception of the case treated with one-half gm. streptomycin daily for 42 days. Although the improvement in this instance was moderate during the 42-day period, and there was some further improvement subsequently, the final result was not satisfactory. However, many patients fail to respond to much larger doses, and we cannot conclude from experience with a single case that the one-half gm. dose of streptomycin given for 42 days would be generally inadequate. It was interesting to note, and this is supported by observations on a much larger number of patients treated for exudative lesions at this hospital, that the frequency of streptomycin administration, whether five times daily, twice daily, or once daily, had no apparent effect on the results.

Generally with *Mycobacterium tuberculosis* not previously exposed to streptomycin, growth will be inhibited by a very low concentration of the drug; usually one microgram or less of streptomycin per milliliter of media. There is growing evidence that we may be dealing with a mixed flora in which organisms sensitive to streptomycin so greatly outnumber those that are resistant that it is quite difficult

to detect the resistant strains unless special methods are employed. After sufficient contact with streptomycin the sensitive bacteria may be inhibited, permitting the resistant strains to flourish until a predominantly resistant population is present in the sputum. Whatever the process underlying production of resistance, once present this resistance is apparently permanent. That resistance is of extreme importance is emphasized by the therapeutic failure of streptomycin in the case described in this report.

Another confusing factor in the problem of resistance is that all patients do not develop resistance at the same rate. Wide variation exists in this, some becoming highly resistant during the first 30 days of treatment, others remaining sensitive during an entire 120-day period. The Streptomycin Committee of the Veterans' Administration has recently reviewed the results obtained in the treatment of a large number of patients with streptomycin on the various doses for 120-day periods. Approximately 70 percent of these patients were streptomycin-resistant by the end of 120 days, whether the daily dose was 2, 1, or ½ gm. Cases 2 and 4, still sensitive after 120 days of treatment, fortunately fall within the 30 percent that do not become resistant. A relatively small percent of the patients treated became resistant during the first 42 days of the 120-day period. It therefore appeared advantageous and therapeutically sound to treat patients for a shorter time and still obtain satisfactory results. Until further data are available, we therefore recommend 1 gm. of streptomycin for 42 days in the treatment of selected cases of recent posthemoptoic spreads.

As demonstrated, dramatic results may be obtained but this emphatically does not mean that all such cases should receive streptomycin. It is common knowledge that a considerable number of posthemoptoic spreads resolve spontaneously on bed rest alone. In our experience, penicillin, administered from 7 to 10 days, may appreciably shorten the initial acute febrile episode so often associated with fresh posthemoptoic lesions, by controlling the hemoptoic component of the lesion. If the infiltration then shows further evidence of resolution, and is not too extensive, streptomycin might not be indicated. However, progression may take place, with or without penicillin, or the new lesion, extensive in nature, may remain stationary. The decision then, as to whether streptomycin should be given, will depend upon circumstances of the individual case. If the drug is to be used, treatment should not be deferred too long, since the more recent the tuberculous exudative component, the more effective streptomycin will be. The course of the disease can generally be determined within 2 months of the acute hemoptoic spreads. In the so-called "panic" case, with massive posthemoptoic spread and

fulminating illness where time is a vital factor, streptomycin given promptly, possibly combined with penicillin for the first 2 weeks, may constitute the major factor in the patient's ultimate recovery, provided that bacterial resistance is not present initially.

CONCLUSIONS

1. Streptomycin is an effective agent in treating the tuberculous component of recent posthemoptoic spreads, provided the tubercle bacilli are sensitive.

2. The two-gm. daily dose is too toxic and has no advantage over one-gm. or one-half-gm. daily dose.

3. Streptomycin may be administered in a single daily intramuscular injection without loss of therapeutic efficacy.

4. The 120-day regimen is apparently too long a period of treatment. Evidence to indicate that 42 days may be sufficient is presented.

5. The 42-day period of treatment, with daily dose of 1 gm. in a single injection is suggested, as bacterial resistance generally does not develop during this period. In case of relapse, retreatment is more likely to be effective.

866046°—50——2

"Painful Feet" in American Prisoners of War

ROBERT B. LEWIS, *Lieutenant Colonel, U. S. A. F. (MC)*[1]

A LARGE number of Americans who were prisoners of war in the Philippines suffered from a bizarre disease in which the principal symptom was "pain in the feet." The disease was undoubtedly due to dietary deficiencies but differed from classical beriberi.

HISTORY

Several authors quoted by Labernadie (*1*) described similar syndromes; one occurred among soldiers on a deficient diet while on campaigns in Europe during the latter part of the eighteenth and early part of the nineteenth centuries. A pseudo-epidemic occurred in 1854 among troops in Crimea; a second occurred during famines following wars; and a third, under the name acrodynia raged through an old people's asylum and among the population around Paris. In Indochina the expression "burning of the feet" has become current in the medical vocabulary and one finds reference to it frequently in the Annual Health Service Reports of Pondichéry; officers who had been in charge of the prisons in 1818 had often observed these cases of burning feet and believed the cause to be the dietary regime. Labernadie described one of his own cases who had almost identical symptoms and signs as those which occurred in American war prisoners; the patient had a positive blood Wassermann test and because antisyphilitic treatment resulted in a favorable response he concluded that the burning of the feet was syphilitic in origin.

A Japanese physician reported to the author that Nipponese troops around Hong Kong had had similar symptoms which the Japanese physicians thought to be caused by cold weather. This same physician had examined the literature in the Bureau of Science in Manila and found three articles describing a similar syndrome occurring in Malaya, British Somaliland, and British Guiana. In one of

[1] U. S. Air Force School of Aviation Medicine, Randolph Air Force Base, Tex.

these locations the painful burning feet were noted to occur in female Chinese laborers but not in male Chinese laborers. It was observed that the women washed their rice prior to cooking and threw away the washings, but the men cooked their rice unwashed. It was concluded from this that there was a vitamin B deficiency in the rice eaten by the women. However, the disease was not believed to be classical beriberi.

A painful feet syndrome, apparently identical with that being described, occurred in other allied prisoners of World War II in the Far East (*2*) (*3*) (*5*). Burgess (*4*) described neuritis and edema among British prisoners in Singapore but did not give a detailed clinical picture of the condition.

INCIDENCE

The present discussion is based upon observations made of the disease when it first appeared at Cabanatuan prison camp on Luzon, in September 1942, 10 months after the beginning of the war and 6 months after the beginning of imprisonment. Four months following the appearance of the syndrome, and at the time of its greatest incidence, of 2,600 patients admitted to the hospital for all causes 75 percent complained of painful feet. The incidence among the remainder of the camp population was also high, although no accurate data are available. Medical officers reported a large number of cases in other prison camps in the Philippines.

NUTRITIONAL BACKGROUND

The following description of the dietary situation is the basis for assuming that the primary etiologic factor in this disease was dietary deficiency.

Prior to their arrival at Cabanatuan prison camp on 1 June 1942, American prisoners had already experienced starvation and often illness for 1 or 2 months, depending on whether they were captured on Bataan or Corregidor. Also, during the defense of these areas most troops were placed on one-third field rations, which consisted principally of polished rice, and at times small amounts of canned meat, fish, or fresh carabao meat. Many persons, especially in the forward areas, had received even less food and before the capitulation of Bataan on 9 April 1942, a few cases of nutritional edema had been seen. On arrival at Cabanatuan early in June 1942 a number of the prisoners had nutritional edema.

From 1 June 1942 to November 1942, the diet given the prisoners contained only 800 to 1,000 calories per man per day, and consisted almost entirely of polished rice and a green leafy swamp weed, kangkong. A few gourds and native squash had been issued before kangkong was made available. At times, yellow or white corn was

added to the diet in lieu of kangkong. The prisoners received no meat. The condition of painful feet first appeared during this period. The few individuals who had money during these first several months were able to buy extra food in the commissary, such as canned meat and fish, peanuts, beans, bananas, oranges, limes, sugar, and coconuts. Very few had money after a month or two and the remainder had to depend entirely on the Japanese-issue diet. It is interesting to note that more than one-third of all money spent by prisoners during this desperate period of starvation was used to buy tobacco.

Beginning in December 1942, and for approximately the next year, the diet furnished by the Japanese was supplemented by Red Cross food supplies [1] and food purchased in the commissary. This diet contained 2,000 to 3,000 calories per day, largely in the form of carbohydrate foods such as polished rice, camotes, casaba, gabi, or corn. During this period approximately 50 gm. of carabao meat were issued daily per man by the Japanese, and additional carabao meat was purchased through the commissary from welfare and private funds. There was a scarcity of fat in the diet. The Japanese maintained that they were issuing 20 gm. of lard substitute or coconut oil per man per day, but in general, this figure exceeded the amount actually received.

It was difficult to determine the vitamin content of the diet. The polished rice was dirty and contained so many bugs, rocks, and worms that repeated washings were required. There is a possibility that a large part of any B_1 remaining in the aleurone layer of the kernels after milling, was thrown away with the washings. The green vegetables in the form of squash, okra, eggplant, tilinum, camote tops, and radish tops contained some B_1, but they were boiled for a considerable time, thus destroying much of the thiamine, even though it is known to be quite heat resistant. In addition, a number of persons insisted on discarding the water in which the vegetables were cooked. The nicotinic acid content of the diet was almost negligible except for those who could afford to buy supplementary foods, such as peanuts. Vitamin C was received in adequate quantities in the form of limes, tomatoes, oranges, and pomalos. However, a few cases of clinical scurvy did occur. A few eggs purchased from the welfare fund for the needy, carotenes in the yellow corn, and green leaves of kangkong, tilinum, camote tops, and radish tops were the only known sources of vitamin A in the diet. Beginning in November 1942 about two carabao livers per day were available for the sick. After several months of imprisonment, a limited amount of cod liver oil was available for treatment of cases with signs of vitamin A deficiency. However, in September 1942 when cases of xerophthalmia suddenly oc-

[1] Each man received 2½ 11-pound boxes of canned food, and in addition there was considerable bulk food issued through the general mess from the Red Cross shipment.

curred in large number, the supply of cod liver oil was so meager that only enough could be spared to instill a few drops into the conjunctival sacs of patients with corneal involvement.

The diet again became poor in December 1943, but fortunately another shipment of Red Cross foods and medicine arrived. Each man received 4⅛ 11-pound boxes of food from this shipment. It appeared as if the Japanese deliberately cut the issued rations when it became known that American Red Cross food was on its way to the camp. The Red Cross food lasted 3 or 4 months, after which time the prisoners had to depend almost entirely on the Japanese-issue diet, which at this time approached in quality and quantity the diet received during the first 6 months of imprisonment.

The deficiencies in the diet were aggravated by other conditions prevalent in the camp. Because of dysentery or diarrhea, many patients derived very little benefit from their already skimpy diets, and in addition, those with untreated active malaria, suffered from a loss of appetite. Another factor which may have led to the loss of water-soluble vitamins was the unexplained diuresis, especially nocturnal, which was experienced by almost everyone.

PATHOLOGY

Unfortunately, the Japanese would not permit autopsies to be performed on Americans who died in the prison camp until February 1943. At this time the death rate was only three or four per month, and only one man with a history of painful feet was known to have died after this date. His painful feet had occurred several months previously and were clinically cured at the time of his death. At autopsy nothing suggestive of vitamin deficiency was visible grossly. There was no enlargement of the heart and no excess fluid in serous cavities.

SYMPTOMS AND SIGNS

In general, the disease followed a more or less characteristic pattern. Usually the first symptom was a slight aching in the balls of the feet, unaffected by walking or movement of the joints of the feet. However, fear that motion would increase the discomfort led to a tendency to favor the feet in walking. For example, in walking down stairs, the patients would side-step down and often steady themselves by placing a hand on the edge of the doorway rather than descending a step at a time. The pain which has a prominent burning component, gradually increased in severity, and was described as a constant burning ache across the balls of the feet, being more severe in the region of the metatarsophalangeal joint of the big toe. The pain was continuous and definitely interfered with sleep. The discomfort was

described as deep, seemingly in the bones, and definitely not in the skin. Many patients would walk around most of the night feeling that it was useless to attempt to sleep. Others complained that the pain was so severe that they feared they would become insane. Still others evidenced emotional instability manifested by crying spells. Words hardly suffice to describe the agony experienced by men afflicted by this syndrome. When reclining they struck a characteristic pose lying on their backs, their feet in the air with the thighs and knees flexed. A characteristic gait was developed in which the knees were lifted somewhat higher than usual and the legs were lifted but not extended in stepping, so that the legs were practically perpendicular to the ground. By this means the feet always struck the ground flat, resulting in a springless gait and shifting of the body laterally to the side of the foot striking the ground to lessen the impact. As motion of the foot joints did not increase the pain, no reason could be ascribed for this gait. Occasionally in very severe cases similar pain involved the hands.

Shortly after the onset of the burning, aching pains in the balls of the feet, a second type of pain occurred, characterized by sharp excruciating shooting pains arising in the vicinity of the metatarsophalangeal joints, especially the first and second. At times in very severe cases, these pains also radiated up the legs. If the hands were involved, the pains sometimes radiated up the arms. These pains were of short duration, lasting only a few seconds, but making their appearance at any time of the day or night. They were described to be very much like severe electrical shocks and often of such severity as to cause the afflicted to jump and cry out.

Hyperesthesia of the skin of the feet was always present in varying degrees, being most severe on the dorsal surfaces of the toes, and also involving the soles of the feet in severe cases. Hypersensitivity was so intense in most cases that it made the weight of bed clothes unbearable. Scratching or touching the toes or soles of the feet would cause many patients to jump and scream violently. It was cruel to perform a Babinski test in these cases. Persons complaining of hyperesthesia of the skin of the soles of the feet developed a different mode of navigation. In order to reduce the contact area of the extremely hypersensitive plantar surfaces, these wretched patients hobbled around only when absolutely necessary and then on the external margins of the feet.

Peculiar dull red mottling of the skin of the feet, especially in the distal portions, was noted in almost all cases. This mottling was blanched by digital pressure and was accompanied by a superficial desquamation of the epithelium. Some noted hyperhidrosis of the feet. In some patients edema of the feet and legs was present, with no apparent relationship between edema and the development of the

painful feet syndrome. Many patients in the camp who developed edema did not become afflicted with painful feet and vice versa. In June, July, and August of 1942, the edema was very frequent and severe, whereas the painful feet first appeared the following September. The edema might possibly be explained as resulting from a deficiency of protein in the diet. However, Keys (7) has warned against such a simple explanation of famine edema and has described its occurrence in starvation with normal plasma protein levels. (Unfortunately, we had no laboratory facilities to perform blood chemistry determinations.) Absence of other signs of heart failure made the possibility of cardiac edema unlikely. As a rule, patients with extensive dependent edema were not bedfast, and experienced no difficulty in getting around except for the unwieldiness of their legs caused by the increased weight. The edema of the feet often stretched the dorsal skin to the breaking point, resulting in large weeping ulcerations. It was not unusual for patients, with or without painful feet, but with considerable edema, to spontaneously have extreme diuresis and lose all their edema in a day or two. Shortly thereafter they would complain of the appearance of the pains of the feet. On the other hand, some patients with painful feet without edema would subsequently have edema, at which time the pain would cease or become less severe, only to reappear when the edema was reduced.

Cardiac and respiratory symptoms were rare, but a few persons complained of precordial pain and tachycardia. Others said they felt their hearts were turning over. Examination of these persons showed a tachycardia of 120 to 150 per minute with numerous extrasystoles. There were no electrocardiographic or roentgenographic machines available. During the period that the condition of painful feet was prevalent a few sudden deaths occurred in young adults for which there was no obvious cause. Some of these may have been the result of cardiac involvement.

It is interesting that motor weakness or paralysis rarely occurred and then in only very severe and long-standing cases. Muscle atrophy did not occur except as a part of the general tissue loss from starvation. Muscular contractures did not develop in any of the cases, as was to be expected, because of the rarity of muscle paresis or paralysis. A few persons did develop footdrop, but it was impossible to determine whether this resulted from a dietary deficiency or was the result of a diphtheria epidemic which occurred in late 1942. (Before the fall of Bataan, three cases of peroneal nerve palsy with footdrop were seen, but they were not believed to have any relation to the condition being described because they came on so much earlier and were unaccompanied by sensory disturbances.) Two patients showed paralysis of leg muscles, but both had diphtheria in July or August 1942 prior to

the development of the paralysis. The deep reflexes of the knee and ankle were normal, except in the few severe and prolonged cases where muscle weakness or paralysis was present. There were no other demonstrable abnormal reflexes. Loss of vibratory sense over the malleoli was common. Deep tenderness of the muscles of the feet or legs was infrequently present. Partial anesthesia of the involved parts was the rule, and in very severe cases, the anesthesia was complete. The legs, thighs, external genitalia, and abdomen were anesthetic in a few severe cases.

A small percentage of patients had amblyopia and an ophthalmoscopic examination showed optic atrophy. Visual fields were done on a majority of these patients with an eye involvement, revealing contraction of varying degrees; the ophthalmologist in the camp stated that he believed that 25 percent had organic eye involvement. Others, including the author, believe this estimate to be too high. The ophthalmological findings have been described by Bloom et al. (*8*).

LABORATORY EXAMINATIONS

Facilities were not available to perform blood chemistry examinations. Hemoglobin content, red and white cell counts, and differential counts were done on the 115 severely ill patients in the experimental group. The only hematologic abnormality noted was slight secondary anemia present in most of these. No abnormalities were found in routine urine examinations. .

TREATMENT, COURSE, AND PROGNOSIS

No treatment was available not even analgesics, during the first 3 months of the disease. The patients tried a variety of self-administered remedies, such as massaging the feet or immersing them in hot or cold water, but none of the procedures were beneficial and heat definitely aggravated the pain.

It was decided to attempt injections of normal saline (30 to 40 cc.) into the sacral canal because the suffering of the afflicted was so great. (Injections of amounts sufficient to cause local discomfort had been used with some success in cases of sciatica.) Several patients stated they obtained relief from the pain immediately following injections and remained free for several days. However, optimism about this form of treatment was cut short, when a patient volunteered the information that the pain in his hands was also cured. This incident raised the question as to whether or not a psychogenic factor was a part of the syndrome. A few patients with painful feet received quinine therapy for malaria and stated that while under treatment the pain in their feet became less severe. Quinine was given to some

patients with painful feet who did not have malaria. Some patients said their pain was less severe from such therapy but others did not improve.

Based on the assumption that the disease might be caused by a water-soluble vitamin deficiency, cultures of yeast were made in a thin watery rice gruel in an effort to obtain some vitamin B. Patients were given a few tablespoonfuls each day without definite results. Obviously very minute quantities of vitamins could be obtained by this crude method.

The Japanese decided to investigate the conditions at Cabanatuan prison camp, about 1 February 1943, approximately 5 months after the appearance of the disease. A Japanese physician, formerly an instructor in Physiology at the University of Mukden, was sent to the camp and under his direction a board of 8 American medical officers was convened for the study. Approximately 115 of the most severe cases of painful feet, were selected for investigation. Some were bedridden, or perhaps one should say floor-ridden because of suffering from pain and starvation. Patients were divided into groups of 10 to 15. One group, used as controls, received no treatment except placebos, a second group received liver extract, a third thiamine chloride, a fourth an injectable form of inorganic phosphate, a fifth vitamin C, a sixth nicotinic acid, a seventh injections of normal saline into the sacral canal, and an eighth all the treatments given to groups two to seven, inclusive. The Japanese physician told the board what drugs and medicine he had available and those which were to be used. It was obvious that some of the medicine to be used would probably be valueless, but the camp physicians were agreeable in the hopes of obtaining a reasonable supply of thiamine, which, it was believed, was the principal substance needed to prevent or cure the disease. The thiamine chloride furnished by the Japanese was an aqueous solution for subcutaneous injection and contained 1 mg. of thiamine per 2 cc. of solution and in order to obtain an effective dosage a large-volume injection had to be given. This procedure was very uncomfortable for the patients, since they had practically no subcutaneous fat. An arbitrary daily dose of 10 mg. was used since the supply was limited. The author does not recall the doses of ascorbic or nicotinic acid prescribed.

Unfortunately, the experiment was allowed to continue for only 6 or 8 weeks, which was too short a time to obtain definite clinical results. Some of the patients who received thiamine, both in the experiment and among other hospital patients, appeared to improve a little faster than those who did not receive it. Evaluation was further complicated by the fact that the general diet of the camp was improved about 2 months before the experimental study began.

On the improved diet, from December 1942 to December 1943, almost all cases of nutritional edema and painful feet were either cured or notably improved. Limited amounts of thiamine, nicotinic acid, and ascorbic acid were available. However, at the end of the period an appreciable number of persons still complained of painful feet. It was suspected that some were using this complaint dishonestly to keep from working on the farm, which was operated by the American prisoners after 1 December 1942. Nutritional edema again became prevalent shortly after December 1943 and there were numerous recurrences and exacerbations of the painful feet syndrome.

A long-term follow-up of these cases was impossible. However, one patient who had a very severe case of painful feet beginning in October 1942 was observed in the AAF Regional Hospital, Santa Ana, Calif., in January 1946, at which time he still complained of shooting pains and hyperesthesia of the soles of the feet to a minor degree, although the burning aching pain had disappeared. In July 1949 this patient was again observed at Randolph Air Force Base, and he complained of shooting pains in the feet several times daily and dull aching pain in the balls of the feet after walking. Other patients are known to have been hospitalized for recurrences at this late date.

OTHER DEFICIENCY DISEASES AND DEATH RATES

Other nutritional deficiency diseases appeared in the camp population of 8,000 to 9,000 men at about the same time that the painful feet syndrome occurred. Pellagra and xerophthalmia were prevalent, and a few cases of scurvy and a greater number of ariboflavinosis cases were recognized. During June and July 1942 the death rate was about 35 per day, primarily from malaria and dysentery with superimposed starvation. Quinine for treating malaria became available in adequate amounts the latter part of July 1942, reducing the death rate by half. For the succeeding 4 months the death rate remained approximately 16 to 18 per day. The latter part of November 1942 the death rate was again reduced to half because of (*a*) the arrival of Red Cross medicine and food, (*b*) an increase in the Japanese-issue diet, including some protein in the form of carabao meat, and (*c*) the purchase of food for the needy from a welfare fund, made possible by monthly donations of American officers from the pay received from the Japanese beginning in December 1942.

CASE REPORT

A 33-year-old medical officer first noticed a slight, dull, burning, aching pain in the balls of both feet about 1 November 1942. The pain gradually increased in severity, interfered with sleep, and reached a maximum in about 30 days.

Shooting pains and hyperesthesia of the dorsal surfaces of toes was moderately severe. There was dull red mottling of the skin with slight superficial desquamation of the skin over the distal half of the feet. The dorsal surfaces of the toes became hypersensitive, making the weight of bed clothes on the feet unbearable. The knee and ankle jerks were normal and there was no motor paralysis or muscle weakness. Administration of 10 mg. of thiamine subcutaneously every other day was begun 2 weeks after the onset of symptoms and this was continued for 3 months. Approximately 1 month after onset of symptoms the diet was improved. At the end of 3 months the burning, aching pains in the balls of the feet were no longer present; shooting pains only occurred occasionally and were relatively mild. The hyperesthesia of the toes was alleviated considerably but was not eliminated entirely. Mottling and desquamation of the skin persisted. The feet remained clumsy as a result of a sensory disturbance characterized by feeling "dead." There was no interference with ordinary walking, but it was apparent subjectively and objectively that he was having difficulty in maneuvering his feet in trying to run. After the pain disappeared the sensory disturbance persisted for about 3 months.

DISCUSSION

It is difficult to determine the exact etiologic factor in this condition because of the complicated etiologic factors and the numerous diseases superimposed upon the starvation. However, it is presumed that the disease is a result of vitamin deficiencies. The patients showed many abnormalities described by Williams (*6*) in experimentally induced vitamin B_1 deficiency in humans, such as restricted physical activity, depressed mental states, apathy, irritability, generalized weakness, and loss of memory. Keys (*7*) has described many of these same symptoms in starvation with essentially adequate vitamin intake. Neither of these authors mentioned aching pains in the balls of the feet, although Williams (*6*) did describe tenderness of the soles. The mental depression, irritability, and apathy could also be explained by the very unhappy situation of the prisoners. Even very minor requests were refused by the Japanese, who exercised every opportunity to humiliate and harass the prisoners and deliberately starved them slowly to death. Many persons, not suffering from painful feet evidenced the other general symptoms described.

Muscular paresis and paralysis were almost entirely absent in these cases, whereas motor involvement is the rule in classical beriberi. On the other hand, sensory disturbances were much more severe than experienced in beriberi. By an improved diet, there was improvement or cure in all patients with rare exceptions, and the recovery was more rapid when therapeutic doses of thiamine chloride supplemented the diet. Six months after treatment with 500 mg. of thiamine chloride and an improved diet, one medical officer was cured of moderately severe painful feet. From the clinical observations and the response to food and vitamin B, it is believed that the disease is a peripheral

neuritis, primarily of sensory nerves (a few patients did have muscle paralysis late and therefore must be considered to have had polyneuritis). Since the clinical picture differs from that of classical beriberi, it is concluded that a deficiency of vitamin B_1 alone could not account for the disease. Some other deficiency must play a part in the cause. However, since patients who received thiamine improved more rapidly than those not receiving it, the concensus was that the deficiency of vitamin B_1 was the principal factor in the etiology. Page (*2*) is of the opinion that deficiency of one or more elements of the B complex, closely allied to the P–P factor, is responsible for the syndrome. Cruickshank (*3*) also believes that lack of nicotinic acid and possibly riboflavin results in this symptom complex. In pellagrous humans it is known that, although involvement of the central nervous system is the rule, peripheral nerve lesions are rare. It has been suggested that in those cases in which peripheral nerve involvement has been described, there was probably a concurrent vitamin B_1 deficiency.

Labernadie (*1*) believed that syphilis was the etiologic factor in his case, but *Treponema pallidum* can be excluded as the causative agent in the cases described in this paper.

Cold was not a factor in the cases occurring in the Philippines.

Poor appetite was quite common, but can be explained by illness and the quality of the food items. The majority ate all they received and wanted more, but many, although free from disease, were unable to eat the meager diet.

The fact that recurrences of the symptoms are reappearing as long as 7 years after the initial attack, in spite of a balanced diet, indicates that irreparable damage has been done to the nerves or the nerve cells, or that the capacity to assimilate certain essential foods has been impaired.

SUMMARY

A condition of painful feet which occurred in Americans who were prisoners of war in the Philippines and who had been on a deficient diet for approximately 9 months is described. Several deficiency diseases were prevalent, but this syndrome was outstanding and could be easily recognized. This condition has been noted previously in the Orient and always under conditions of inadequate dietary regimes. It is presumed that the syndrome is the result of vitamin deficiencies, and the symptoms and signs are due to a neuritis primarily of sensory nerves. It is believed that the deficiency of thiamine is the principal but not the only factor responsible for the syndrome.

References

1. Labernadie, V. G. F.: Apropos d'un cas de "burning of feet." Far East. A. Trop. Med., Tr. Seventh Congress (1927) 1: 13–16, 1928.
2. Page, J. A.: Painful-feet syndrome among prisoners of war in Far East. Brit. M. J. 2: 260–262, Aug. 24, 1946.
3. Cruickshank, E. K.: Painful feet in prisoners-of-war in Far East; review of 500 cases. Lancet 2: 369–372, Sept. 14, 1946.
4. Burgess, R. C.: Deficiency diseases in prisoners-of-war at Changi, Singapore; February 1942 to August 1945. Lancet 2: 411–418, Sept. 21, 1946.
5. Harrison, G. F.: Nutritional deficiency, painful feet, high blood pressure in Hong Kong. Lancet 1: 961–964, June 29, 1946.
6. Williams, R. D.; Mason, H. L.; Wilder, R. M.; and Smith, B. F.: Observations on induced thiamine (Vitamin B_1) deficiency in man. Arch. Int. Med. 66: 785–799, Oct. 1940.
7. Keys, A.: Human starvation and its consequences. J. Am. Dietet. A. 22: 582–587, July 1946.
8. Bloom, S. M.; Merg, E. H.; and Taylor, W. W.: Nutritional amblyopia in American prisoners of war liberated from Japanese. Am. J. Ophth. 29: 1248–1257, Oct. 1946.

Administration of Procaine Intravenously

IV. In General Surgery and Obstetrics [1]

JESSE F. BROWN, *Captain, MC, U. S. A.*[2]

THE valuable pharmacologic properties of procaine administered intravenously are: elevation of the pain threshold; depression of nerve transmission; relief of smooth muscle spasm; and depression of the myocardial hyperirritability that may arise during anesthesia. It is through recognition of these properties and their proper application that procaine infusion has become a therapeutic procedure with a broad range of usefulness. Its most common use is for the relief of pain. Even small doses of procaine given subcutaneously raise the threshold of pain at distant sites. The general analgesic action of procaine following its subcutaneous injection was measured by Bigelow and Harrison (*1*) and the effect was found to be nearly equivalent to the relief after acetylsalicylic acid; namely, a ceiling rise about 35 percent above the normal threshold value. This action of procaine is greater after its intravenous injection, and the degree of analgesia is proportional to the blood level obtained. It has been used to provide analgesia for burns, while changing surgical dressings, and to control postoperative pain.

In obstetric patients it has been used alone or in combination with other agents for general anesthesia (*2*) (*3*) (*4*) (*5*). In these patients the infusion was started after labor was well established. The rate of administration, which varied from 10 mg. per minute at the onset of labor to 200 mg. per minute to achieve total anesthesia for delivery and repair, was regulated according to the severity of the pains. Throughout the second stage the rate was slow, but for delivery and subsequent repair operations it was increased. Labor pains were relieved without disturbance of consciousness. The total dose of procaine ranged from 0.3 to 9.7 gm. Administration was continuous throughout labors that lasted from 35 minutes to 15 hours. Allen (*2*) considered this to be

[1] Parts I and II of this series were printed in the December 1949 issue of The Bulletin of the U. S. Army Medical Department. Part III appeared in the January 1950 issue of the U. S. ARMED FORCES MEDICAL JOURNAL.

[2] Brooke General Hospital, Fort Sam Houston, Tex.

the ideal obstetric analgesic since it (*a*) offers convenient, flexible, and rapidly controllable analgesia; (*b*) does not inhibit uterine contractions, and even appears to hasten delivery; (*c*) is apparently harmless to mother and child; (*d*) is free from the depressant action of narcotics and barbiturates on mother and child; and (*e*) is not associated with headache, nausea, disorientation, or other uncomfortable sequelae. Recovery is almost immediate. The procedure is not, however, without danger. In some persons the anesthetic and convulsive doses are nearly the same. Therefore certain women cannot be given complete analgesia without danger of convulsions unless a second drug is given to depress the central nervous system.

Procaine infusion has also been used for surgical anesthesia. Scattered reports indicate that it gives anesthesia suitable for most minor procedures and for major procedures which do not require muscular relaxation. Graubard and Peterson (*6*) used it successfully for repair of lacerations, but found it unsatisfactory for the reduction of fractures. Trifari and Martin (*7*) have used it in combination with curare for minor plastic surgery and in abdominal surgery. They summarized the advantages to be gained from intravenous procaine anesthesia and recommended its continued trial. Its growing popularity is based on the ease and simplicty of administration, its freedom from undesirable side effects, and the prolonged analgesia following its use. Postoperative respiratory depression, vomiting, and malaise, which are so commonly seen after morphine or other types of general anesthesia are eliminated. With procaine control of pain generally outlasts therapeutic doses of morphine. Finally, postoperative care is simple because consciousness is not affected unless large amounts are used and recovery of consciousness is rapid. In spite of these advantages procaine infusion has not been widely adopted for general anesthesia. Blood levels of procaine which are necessary to achieve unconsciousness are on the borderline of toxicity and untoward reactions are feared. Allen and Safford (*8*) have stated: "While retaining procaine for intravenous analgesia, we have abandoned it for general surgical anesthesia, because of rare unpredictable collapse without convulsions and without known means of prevention."

Another application of the general analgesic property of procaine is its combination with other agents for general anesthesia. It has been suggested that by depressing the central nervous system with other general anesthetic agents, the likelihood of convulsions is reduced and the margin of safety is thereby widened (*9*). Although all central nervous system depressants undoubtedly will provide some degree of protection, the barbiturates are specific in this action (*10*). The most practical combination has been found to be pentothal-procaine. With these drugs the hypnotic action of one supplements the

analgesic action of the other. The advantage to be gained from elevating the pain threshold during pentothal anesthesia by a drug that does not depress respiration is apparent. Knight (*12*) (*13*) prefers the fractional dosage technique of administration, using the ratio of 25 mg. pentothal, 10 mg. of procaine, and 10 units of intocostrin. Nitrous oxide and oxygen are administered as soon as the patient loses consciousness. Fraser (*13*) and Fraser and Kraft (*14*) have reported over 1,500 cases without mishap. They balanced the use of pentothal-procaine with various other forms of anesthesia according to the requirements of relaxation. Their technique is a continuous drip of 0.1 to 0.2 percent pentothal with 0.05 to 0.1 percent procaine. For postoperative sedation the ratio of pentothal and procaine is reversed. In all types of operation where relaxation is not an essential requirement, they found pentothal-procaine an excellent combination of anesthetic agents. Our own experience in a few well-selected cases has confirmed its utility.

An example of the use of the sympatholytic property of procaine infusion is found in the treatment of gynecologic disorders. Allen (*2*) suggested its use for treatment of threatened abortion. No report of its use in this condition has been seen. Its use has been suggested for relaxation of the pathologic contraction ring of Bandl and in dystocia. At the University of Texas procaine infusions have been used in treating a large series of patients with menstrual cramps (*15*).

The use of intravenous procaine as a cardiac depressant during general anesthesia is of special interest (*16*). It is paradoxical that all of the commonly known inhalation agents have a depressant effect on the central nervous system but produce a hyperirritability of the heart muscle and cardiac conduction system as a result of which mechanical, chemical, and reflex stimuli may provoke a variety of arrhythmias, some of which may threaten life. Arrhythmias are common with all anesthetic agents. Although the abnormal rhythm often consists of premature contractions or displacements of the pacemaker, the more serious arrhythmias occur more frequently than is usually suspected. Burstein (*17*) has suggested that anesthesiologists use direct-reading electrocardiography as a means of detecting serious disorders in cardiac rhythm that may otherwise pass unnoticed. Because of the diversity of causes of cardiac irregularities, it is not reasonable to expect one form of treatment to correct them all. Stimuli which are capable of disturbing cardiac rhythm may arise from many sources (*15*). When possible, the precipitating causes should be avoided, or otherwise removed as in the observance of frequent rest periods in operations on the heart. These measures do not, however, reverse the underlying cardiac hyperirritability caused by the anesthetic agent.

Attempts to overcome cardiac hyperirritability by the use of procaine stem from the work of Beck and Mautz (*18*) who applied procaine solution topically to the heart and pericardium in operations on the heart. This was found to prevent extrasystoles caused by surface stimuli, to abolish auricular fibrillation when applied over the auricles; and to prevent ventricular fibrillation. Methods of counteracting cardiac hyperirritability went through a period of laboratory development during which the effects of procaine infusion were compared with those of other drugs. Procaine infusion was first used to control cardiac rhythm in anesthetized human patients by Burstein. Following the success of this method he used it prophylactically before inducing anesthesia. Of over 500 patients so treated none with a regular cardiac rhythm developed arrhythmias during operation and none with abnormal rhythm preoperatively showed any increase in arrhythmia.

The experimental work that preceded the clinical application of procaine infusion for the control of cardiac rhythm is extensive. The method used was to sensitize the heart of the experimental animal to chloroform or cyclopropane and then produce arrhythmias by injecting epinephrine intravenously. Drugs being tested were injected in an attempt to stop the arrhythmia, or were given prophylactically to prevent it. Burstein (*19*) believed he could successfully treat ventricular fibrillation produced in this manner by subsequently injecting procaine intravenously. Stutzman, Allen, and Orth (*20*) reproduced his results, but disagreed on their interpretation, when electrocardiograms showed a rapid ventricular tachycardia. Allen, Stutzman, Slocum, and Orth (*21*) pointed out that the essential conditions necessary for the production of cardiac arrhythmias by this method are the presence of the anesthetic agent and epinephrine in the heart and an intact sympathetic pathway between the heart and a brain center above the pons (*21*). They tested a series of drugs of known pharmacologic action and showed that the arrhythmias might be abolished or inhibited at several sites of action. Procaine, carbon dioxide, and quinidine gave protection because of myocardial depression; F883 and ergotamine, by their sympatholytic action; yohimbine, through its adrenolytic action; and morphine by producing either functional decerebration or myocardial depression. Effective amounts of the two most practical drugs that completely protected the heart were 16 mg. in the case of procaine and 15 mg. in the case of quinidine per kilogram of body weight. The effective dose of morphine was 8 mg. per kilogram. Investigation of this problem is progressing with priscoline, dibenamine, and other new drugs.

Procaine infusion has been shown to be both effective and safe for control of the heart rhythm during surgical anesthesia. Two recent

866046°—50——3

articles have described in detail the effect of procaine on the heart. Uhley (*22*) gave 50 mg. per kilogram of body weight of procaine to dogs maintained under sodium pentothal anesthesia. The resulting changes in the electrocardiograms consisted of prolongation of the PR and QRS intervals signifying a mild degree of auriculoventricular and interventricular block. There were also changes in QRS, T-S, and T configurations, suggesting some alteration in the depolarization and repolarization patterns. Long (*10*) injected varying doses of procaine (4 to 80 mg. per kilogram of body weight) rapidly into dogs anesthetized with sodium pentobarbital and made frequent estimations of the blood concentration of procaine as well as continuous electrocardiographic tracings. In this manner electrocardiographic changes were correlated with blood concentrations of procaine. No changes occurred in the electrocardiographic pattern, heart sounds, or blood pressure when the dose was 4 mg. per kilogram of body weight, but as the dose was increased a series of progressive changes occurred that were maximal when the blood level was at its peak and regressed as the blood level fell. All of the electrocardiographic changes were reversible and returned to the control state in from 1 to 3 minutes. Above a dose of 50 to 60 mg. per kilogram of body weight there was a tendency for arrhythmia to occur, and at 60 to 80 mg. per kilogram of body weight, 3 out of 20 dogs developed ventricular fibrillation. Some evidence was presented that damaged heart muscles are more sensitive to procaine than are normal heart muscles. The significance of this fact, if confirmed, deserves further exposition because it implies that clinical response is best where the need for treatment is greatest.

Although doses comparable to those used in animal experimentation are never used clinically, improvement in abnormal cardiac rhythm is often experienced following procaine infusions. The mode of action of procaine is believed to be the summation of several effects (*23*). Electrocardiographic studies have shown that procaine can cause a displaced pacemaker to revert to the sinus node. By depressing activity of the extrinsic cardiac nerves, it reduces extracardiac nervous stimulation. Suppression of the adrenal medullary secretion prevents reflex liberation of epinephrine. Conduction through the bundle of His and the ventricular musculature is slowed and, to a lesser extent, conduction is slowed through the auriculoventricular node and possibly through the atrial musculature. The refractory period after systole is prolonged, and the excitability of the myocardium to electric stimulation is diminished. The result of these combined actions is decreased disturbance of the cardiac mechanism from a variety of insults which arise during operation.

Because of the rapidity with which procaine is destroyed after intravenous injection, the use of a single injection before inducing

anesthesia to serve as a prophylactic agent against cardiac dysfunction seems illogical. When, however, disturbance in rhythm occurs during anesthesia because of stimuli arising from surgery, a single injection is frequently adequate to restore normal rhythm, particularly if the stimuli are thereafter discontinued. Burstein originally recommended 50 mg. but has recently advocated a single dose of 100 mg. That this dose is not excessive is seen from the reports of two patients who inadvertently received 1 gm. intravenously without ill effect when 100 mg. was intended. The author gives a continuous infusion of a 0.2 percent solution slowly throughout an operation with an increased rate at times of increased stimuli or when arrhythmias are detected. Procaine infusion is given regularly to patients undergoing thoracic operations and to patients who are poor surgical risks. Other patients are given single injections of procaine when it seems indicated. In using procaine prophylactically it is difficult to judge the results except in comparison with the expected course of the patient.

CONCLUSIONS

Procaine infusion is a valuable means of relieving pain and muscle spasm. In combination with other agents it may be used as an anesthetic. It is a means of preventing and correcting disturbances in cardiac rhythm during anesthesia. Its availability does not relieve the surgeon of his obligation to handle tissues gently nor the anesthetist of his responsibility to practice his art according to the best principles. It does, however, add to the latter's armamentarium a procedure that further contributes to the safety of anesthesia, particularly in patients who are poor surgical risks.

REFERENCES

1. BIGELOW, N., and HARRISON, I.: General analgesic effects of procaine. J. Pharmacol & Exper. Therap. **81**: 368–373, Aug. 1944.
2. ALLEN, F. M.: Intravenous obstetrical anesthesia; preliminary report. Am. J. Surg. **70**: 283–290, Dec. 1945.
3. JOHNSON, K., and GILBERT, C. R. A.: Intravenous procaine for obstetrical anesthesia. Anesth. & Analg. **25**: 133–146, July–Aug. 1946.
4. Personal communication from Dr. John W. Winter.
5. SALVINI, A., and FERRERO, F.: Intravenous "Recorcain" (procaine) analgesia in labor. Riv. ital. diginec. **30**: 397–407, 1947, Abstr.: J. Obst. & Gynaec. Brit. Emp. **55**: 830, Dec. 1948.
6. GRAUBARD, D. J., and PETERSON, M. C.: Therapeutic uses of intravenous procaine. Anesthesiology **10**: 175–187, March 1949.
7. TRIFARI, L. M., and MARTIN, S. J.: Civilian and military aspects of intravenous anesthesia. Anesth. & Analg. **26**: 45–63, March–April 1947.
8. ALLEN, F. M., and SAFFORD, F. K.: Animal experiments with procaine and related drugs. Footnote, Anesth. & Analg. **27**: 121–136, May–June 1948.
9. BURSTEIN, C. L.: Misuse of adrenalin and coramine. Am. J. Surg. **73**: 102–103, July 1949.

10. LONG, J. H.; OPPENHEIMER, M. J.; WESTER, M. R.; and DURANT, T. M.: Effect of intravenous procaine on heart. Anesthesiology **10**: 406–415, July 1949.
11. KNIGHT, R. T.: Present developments in combined anesthesia. J. Michigan M. Soc. 46: 90–91, Jan. 1947.
12. KNIGHT, R. T.: Curare in anesthesia. J. Indiana M. A. **40**: 125, Feb. 1947.
13. FRASER, R. J.: Intravenous pentothal-procaine analgesia. Anesth. & Analg. **27**: 159–163, May–June 1948.
14. FRASER, R. J., and KRAFT, K.: Pentothal-procaine analgesia. Anesth. & Analg. **27**: 282–286, Sept.–Oct. 1948.
15. SLOCUM, C. H.: Personal communication.
16. BURSTEIN, C. L.: Treatment of acute arrhythmias during anesthesia by intravenous procaine. Anesthesiology **7**: 113–121, March 1946.
17. BURSTEIN, C. L.: Utility of intravenous procaine in anesthetic management of cardiac disturbances. Anesthesiology **10**: 133–144, March 1949.
18. BECK, C. S., and MAUTZ, F. R.: Control of heart beat by surgeon, with special reference to ventricular fibrillation occurring during operation. Ann. Surg. **106**: 525–537, Oct. 1937.
19. BURSTEIN, C. L.; MARANGONI, B. A.; DEGRAFF, A. C.; and ROVENSTINE, E. A.: Laboratory studies on prophylaxis and treatment of ventricular fibrillation induced by epinephrine during cyclopropane anesthesia. Anesthesiology **1**: 167–186, Sept. 1940.
20. STUTZMAN, J. W.; ALLEN, C. R.; and ORTH, O. S.: Failure of procaine to reverse cyclopropane-epinephrine ventricular fibrillation. Anesthesiology **6**: 57–60, Jan. 1945.
21. ALLEN, C. R.; STUTZMAN, J. W.; SLOCUM, H. C.; and ORTH, O. S.: Protection from cyclopropane-epinephrine tachycardia by various drugs. Anesthesiology **2**: 503–514, Sept. 1941.
22. UHLEY, M. H., and WILBURNE, M.: Effect of intravenous procaine on electrocardiogram of dog. Am. Heart J. **36**: 576–581, Oct. 1948.
23. RITTRICH, N. M., and POWERS, W. F.: Intravenous procaine in thoracic surgery. Anesth. & Analg. **27**: 181–196, July–Aug. 1948.

Recent Advances in Plastic Surgery

GORDON S. LETTERMAN, *M. D.*[1]
WILLIAM C. MELOY, *M. D.*[1]

THE increasing importance of and the medical profession's rapidly growing interest in plastic and reconstructive surgery, is indicated by the many papers on this subject which have appeared in recent months. This article is a brief review of a few of the more significant advances in this specialty made during the last 2 years. Those topics of practical interest to the military surgeon are featured. More comprehensive and detailed reviews of the recent literature have been written by Letterman and Brown (*1*) (*2*) and Letterman and Meloy (*3*).

COMPOSITE GRAFT

Brown, Cannon, et al. (*4*) utilize a free composite graft obtained from the ear and composed of two surfaces of skin and intervening cartilage, for the repair of nasal defects. In a single operation a comparatively large bulk, which is ideally suited for the repair and reconstruction of free tissue of the columella, ala, and tip of the nose, is transferred. Restorations with the composite graft match the nasal border in texture, contour, color, and appearance. The defect of the ear is easily repaired by direct closure or local flap. Armstrong (*5*) employs composite grafts in the reconstruction of full thickness losses of the lower eyelid. This type of graft undoubtedly will prove advantageous in the restoration of other facial features.

MANDIBLE

Byars (*6*) (*7*) deals with the preservation and restoration of mandibular function and contour. An internal bar fixation method is described which meets the immediate postoperative needs following mandibular resection. It maintains the fragments of the mandible in their original position and therefore in the optimum position for future bony reconstruction. Kanthak and Hamm (*8*), Padgett et al. (*9*), and Dingman (*10*) discuss the correction of mandibular deformities. Blocker (*11*) (*12*) (*13*) emphasizes the importance of bone

[1] Consultant in Plastic Surgery, Army Medical Center and Walter Reed General Hospital, Washington, D. C.

grafting in mandibular reconstruction. He prefers to use iliac bone grafts of pure cancellous bone. An adequate skin covering free of scar tissue and infection is a prerequisite for repair. Delk and Letterman (*14*) use a combination of two techniques, internal wire fixation with the Kirschner type pin and an arch bar of the Risdon variety, in the management of complicated fractures of the symphysis. Barclay et al. (*15*) analyze the care and treatment of the fractures of the upper and lower jaws acquired by members of the Canadian Army in World War II.

SKIN GRAFTS AND PEDICLE FLAPS

Kernwein (*17*) finds recovery of sensation in split thickness grafts is patchy and never complete. The fine degree of sensation exhibited by pedicle flaps is never found in the free split graft. Baxter and Entin (*18*) state that free split thickness skin grafts may be stored at temperatures between 8° and 0° C. for 3 weeks and still take. Brown (*16*) writes that in the Medical Department report of World War I some 300 pages were devoted to plastic surgery without any mention of free skin grafting except for one sentence in which a small oral graft was suggested. In contrast to this, about one-third of all the reconstructive surgery done in World War II consisted of treatment of wounds by free skin grafting. Bunnell et al. (*19*) recognize a new entity in the hand consisting of a fibrous contracture of the intrinsic muscles. The pathological changes are similar to those observed in Volkmann's ischemic contracture. Clifton (*20*) directs attention to unusual innervation of intrinsic muscles by the medial and ulnar nerves. Such exceptional cases are of importance in evaluating nerve injury pre- and post-operatively. Cannon et al. (*21*) (*22*) describe the use of an open jump flap from the abdominal wall to resurface extensive defects and deep scars of the lower extremities. This method of repair decreases the time required for such reconstruction from many months to as few as 10 weeks. Converse (*23*) gives in detail the repair of defects of the extremities with nontubulated pedicle flaps. He believes the repair of lower extremity defects with cross-leg flaps are more satisfactory than a closed carried flap. Letterman (*24*) uses plexiglas splints to distinct advantage in the immobilization of the lower extremities during the attachment of cross-leg flaps. Lewin (*25*) gives the indications for free skin grafts and flaps in the repair of compound injuries of the lower extremities.

EARS

Brown, Cannon, et al. (*26*) present a simplified method for restoration of the external ear. The scarred edges of the remaining ear stump are excised. These freshened edges are in turn implanted in

a scalp flap raised above and behind the ear. Several weeks later a piece of preserved cartilage carved to resemble the ear is inserted under the flap. After all incisions are well healed and the edema has subsided, the cartilage and covering flap are dissected free of the skull. A soft tissue layer is left covering both surfaces; special care is taken not to expose the cartilage. The two raw surfaces are immediately covered with a split thickness graft. Aufricht (*27*), Conway et al. (*28*), and Peer (*29*) have made notable recent contributions in auricular reconstruction.

EYELIDS AND ORBIT

Schuessler and Filmer (*31*) find a free hairbearing graft taken vertical to and through the eyebrow to have definite advantages in restoration of the cilia. Cannon (*30*) directs attention to the decided superiority of full thickness grafts obtained from the base of the neck for resurfacing eyelids. The platysma muscle in the subcutaneous tissue of the clavicular area apparently imparts special qualities to this skin which make it excellent for eyelid reconstructions. A full thickness graft has good color and there is minimal shrinkage. Spaeth (*32*) divides upper eyelid repair into two major divisions: reconstruction in the presence of a functioning eyeball; and when the socket is surgically anophthalmic. The problems peculiar to each are discussed. Sherman (*33*) stresses the importance of the early reduction of fractures of the floor of the orbit. Pickrell and Clark (*34*) describe and depict a method of tattooing corneal scars with insoluble pigments. This procedure often proves quite beneficial to patients who are not acceptable for the transplant operation. Greear (*35*) reviews the methods used in orbit reconstruction. Buccal mucosa is advocated as the most suitable lining for restored sockets. Callahan (*36*) deals with the repair of canthal deformities.

FACIAL INJURIES AND REPAIRS

Iverson (*39*) stresses the importance of the early care of traumatic tattoos of the face. Vigorous scrubbing of the initial lesion and adequate irrigations will frequently prevent future blemishes. Ordinary carpenter's sandpaper, grit 0 and 00, are used to erase healed tattoo areas. The skin is vigorously abraded, stopping short only of the full thickness. Brown and Cannon (*37*) deal with the salient problems and emphasize the basic principles in the care of patients with facial injuries. Cutting débridement and open packing of facial wounds is never indicated. If severely ulcerated faces are promptly and judiciously repaired, a most satisfactory result can frequently be obtained in one single immediate operation. Indeed, a similar result may not be duplicated by many later procedures. Pressure dressings following injury and plastic repair are as necessary

on the face as for any other part of the body. Brown and Fryer (*38*) emphasize the basic principles in the repair of compound facial injuries. Peacher (*40*) enumerates the speech disturbances resulting from maxillofacial injuries acquired in World War II. Ivy (*41*) has described the repair of healed facial defects with low neck pedicle grafts. Blocker and Weiss (*13*) stress the importance of the patient's personality and adjacent features in reconstruction of facial defects.

SCALP AND CRANIOPLASTY

Peacher, Cannon, and Brown (*42*) evaluate the use of tantalum, bone, and cartilage in the repair of skull defects. Bone is the best material with which to repair the majority of defects. Tantalum is sometimes necessary for the repair of very large defects and in other situations where bone is contra-indicated. Cannon et al. (*43*) stress the necessity of a good viable resurfacing of the scalp prior to the closure of the defect. Kazanjian and Webster (*44*) review the treatment of extensive losses of the scalp.

FACIAL PARALYSIS

Owens (*45*) (*46*) compares the surgical methods available for correction of facial paralysis and is convinced that the procedure of choice is the insertion of fascial strips. Brown and McDowell (*47*) (*48*) use loops of fascia lata connecting the temporal muscle and face in the region of the mouth and columella. Adams (*49*) employs multiple muscle transplantations to correct facial paralysis. None of these workers advise the use of tantalum, wire, or wire mesh.

DECUBITUS ULCER

White and Hamm (*50*) advocate the use of widely undermined flaps of gluteal skin and subcutaneous tissue to cover the defects left by the block excision of the decubitus ulcer. Kostrubala and Greeley (*51*) feel that simple excisions of the entire pyrogenic membrane and flap repair do not remove the immediate cause of bedsores. They stress the importance of removing the bony prominences underlying the ulcer. The complete excision of the ulcer includes a considerable portion of the underlying bone which shows changes ranging from simple fibrosis to chronic osteomyelitis.

SCARS AND KELOIDS

Block and Tsuzuki (*52*) have found a comparatively high incidence of keloids and hypertrophic scars in Japanese sustaining burns from atomic bomb explosions. Considering the treatment of these cases and the incidence of keloid formation among Japanese people, it seems impossible to evaluate the significance of direct ionizing action and

neutron radiation, in the production of these scars. Trusler and Bauer (*53*) believe that application of continued pressure to a new scar and radiation therapy are important prophylactic measures in dealing with the repair of keloids and hypertrophic scars.

GENITALIA

Young and Benjamin (*54*) advocate the use of a one-stage operation for hypospadias repair. A free split graft obtained from the anteromedial surface of the arm is used to form the lining of the tube. This procedure has been found more satisfactory than operations involving flap repairs. Price and Penna (*55*) have studied the effectiveness of drugs in preventing postoperative penile erections. The pre- and post-operative use of stilbesterol is recommended. Davis and Berner (*56*) describe the reconstruction, following total avulsion of the skin from the penis and scrotum.

NOSE

Byars (*57*) describes in detail the correction of the more frequent nasal deformities. Standard rhinoplasty procedures are based upon the planned alteration of the size, shape, and interrelationship of the component nasal parts. The patient's facial features, and even more important, the whole patient must be given careful consideration whenever any change in the nasal structure is contemplated. Steffensen (*58*) reviews the methods for correcting deviations of the anterior-inferior margin of the septum and warns especially about the injudicious use of submucous septal resection. Maliniac (*59*) describes a compressive suspension splint for the management of comminuted nasal fractures. The instrument combines narrowing of the dorsum with forward traction of the depressed fragments. Rubin, Robertson, and Shapiro (*60*) use implants of polyethylene for nasal, chin, and forehead restorations. The substance is chemically inert and does not appear to have an unfavorable effect on body tissues. Safian (*61*) offers an excellent critical and authoritative review of the more recent articles on rhinoplastic surgery.

CLEFT LIPS AND PALATES

Blair and Robinson (*62*) elaborate upon certain facts pertinent to cleft lip closure. The aim of the surgical procedure is to obtain a symmetrical lip, free-breathing, and a natural-appearing nose without suture marks or prominent scars. Blair's repair involves a modified Mirault flap. Brown, McDowell, and Byars (*63*) present the basic surgical principles and describe in detail the procedures involved in double cleft lip repair. Brown and McDowell (*64*) describe their repair of the single cleft lip. Fogh-Anderson (*65*) gives an historical

account of the Stein-Estlander-Abbe operation. Kazanjian (*66*) deals with the common secondary deformities developing in cleft lip and palate cases. Dorrance and Bransfield (*67*) find the "push back" operation necessary in almost every palate case.

REFERENCES

1. LETTERMAN, G. S., and BROWN, J. B.: Review of reconstructive surgery of the face—1947. Laryngoscope **58**: 552–582, June 1948.
2. LETTERMAN, G. S., and BROWN, J. B.: Plastic and Reconstructive Surgery. Cyclopedia of Medicine. F. A. Davis Co., Philadelphia, Pa. 1948.
3. LETTERMAN, G. S., and MELOY, W. C.: Plastic surgery in the United States; review of 1948 literature. (In press.)
4. BROWN, J. B.; CANNON, B.; LISCHER, C. E.; and DAVIS, W. B.: Composite free grafts of skin and cartilage from the ear. **J. A. M. A. 134**: 1295–1296, Aug. 16, 1947.
5. ARMSTRONG, C. J.: Use of composite graft in reconstructive surgery of lower eyelid. Plast. & Reconstruct. Surg. **3**: 79–83, Jan. 1948.
6. BYARS, L. T.: Preservation and restoration of mandibular function and contour. Ann. Surg. **127**: 863–879, May 1948.
7. BYARS, L. T., and MCDOWELL, F.: Preservation of jaw function following surgery and trauma. Surg., Gynec. & Obst. **84**: 870–877, April 1947.
8. KANTHAK, F. F., and HAMM, W. G.: Surgical treatment of growth deformities of mandible. South. Surgeon **14**: 147–153, Feb. 1948.
9. PADGETT, E. C.; ROBINSON, D. W.; and STEPHENSON, K. L.: Ankylosis of the temporomandibular joint. Surgery **24**: 426, 1948.
10. DINGMAN, R. O.: Surgical correction of developmental deformities of the mandible. Plast. & Reconstruct. Surg. **3**: 124–146, March 1948.
11. BLOCKER, T. G., JR.: Principles of reconstruction of defects about the face. Texas State J. Med. **42**: 527–531, Jan. 1947.
12. BLOCKER, T. G., JR.: Cancellous bone grafts to the jaw. Surg., Gynec. & Obst. **84**: 553–561, April 15, 1947.
13. BLOCKER, T. G., JR., and WEISS, L. R.: Use of cancellous bone in repair of defects about the jaws. Ann. Surg. **123**: 622–640, April 1946.
14. DELK, M. B., and LETTERMAN, G. S.: Fractures of mandibular symphysis with report of cases. J. Oral Surg. **6**: 316–323, Oct. 1948.
15. BARCLAY, L. T.; GORDON, S. D.; and CAMPBELL, H.: Management of jaw fractures. Surg., Gynec. & Obst. **84**: 973–978, May 1947.
16. BROWN, J. B.: Closure of surface defects with free skin grafts and with pedicle flaps. Surg., Gynec. & Obst. **84**: 862–865, April 15, 1947.
17. KERNWEIN, G. A.: Recovery of sensation in split thickness skin grafts. Arch. Surg. **56**: 459–474, April 1948.
18. BAXTER, H., and ENTIN, M. A.: Experimental and clinical studies of reduced temperatures in injury and repair in man. III. Direct effect of cooling and freezing on various elements of the human skin. Plast. & Reconstruct. Surg. **3**: 303–334, May 1948.
19. BUNNELL, S.; DOHERTY, E. W.; and CURTIS, R. M.: Ischemic contracture, local, in hand. Plast. & Reconstruct. Surg. **3**: 424–433, July 1948.
20. CLIFTON, E. E.: Unusual innervation of intrinsic muscles of hand by median and ulnar nerve. Surgery **23**: 12–31, Jan. 1948.
21. CANNON, B.; LISCHER, C. E.; and BROWN, J. B.: Open jump flap repairs of lower extremity. Surgery **22**: 335–340, Aug. 1947.

22. CANNON, B.; LISCHER, C. E.; DAVIS, W. B.; CHASKO, S.; MOORE, A.; MURRAY, J. E.; and MCDOWELL, A.: Use of open jump flaps in lower extremity repairs. Plast. & Reconstruct. Surg. **2**: 336–341, July 1947.
23. CONVERSE, J. M.: Plastic repair of extremities by nontubulated pedicle skin flaps. J. Bone & Joint Surg. **30-A**: 163–194, Jan. 1948.
24. LETTERMAN, G. S.: Plexiglas splints; use in pedicle flap attachments. Plast. & Reconstruct. Surg. **3**: 553–559, Sept. 1948.
25. LEWIN, M. L.: Resurfacing procedures in compound injuries of lower extremities. Ann. Surg. **128**: 66–79, July 1948.
26. BROWN, J. B.; CANNON, B.; LISCHER, C.; DAVIS, W. B.; and MOORE, A.: Surgical substitutions for losses of external ear. Surg., Gynec. & Obst. **84**: 192–196, Feb. 1947.
27. AUFRICHT, G.: Total ear reconstruction: preliminary report. Plast. & Reconstruct. Surg. **2**: 297–306, July 1947.
28. CONWAY, H.; NEWMANN, C. G.; GELB, J.; LEVERIDGE, L. L.; and JOSEPH, J. M.: Reconstruction of external ear. Ann. Surg. **128**: 226–239, Aug. 1948.
29. PEER, L. A.: Reconstruction of auricle with diced cartilage grafts in vitallium ear mold. Plast. & Reconstruct. Surg. **3**: 653–666, Nov. 1948.
30. CANNON, B.: Some recent developments in plastic surgery. S. Clin. North America, 1106–1112, Oct. 1947.
31. SCHUESSLER, W. W., and FILMER, G. A.: Method of restoration of cilia of eyelids. Plast. & Reconstruct. Surg. **2**: 345–347, July 1947.
32. SPAETH, E. B.: Reconstruction of upper lid. Surg., Gynec. & Obst. **84**: 804–808, April 15, 1947.
33. SHERMAN, A. E.: Reconstruction of orbital floor defects. Surg., Gynec. & Obst. **84**: 799–803, April 15, 1947.
34. PICKRELL, K. L., and CLARK, E. H.: Tattooing of corneal scars with soluble pigments. Plast. & Reconstruct. Surg. **2**: 44–59, Jan. 1947.
35. GREEAR, J. N., JR.: Rise of buccal mucosa in restoration of orbital socket. Am. J. Ophth. **31**: 445–453, April 1948.
36. CALLAHAN, A.: Reconstruction of canthi. Tr. Am. Acad. Ophth. **52**: 486–496, May–June 1948.
37. BROWN, J. B., and CANNON, B.: Repair of major facial injuries. Ann. Surg. **126**: 624–632, Oct. 1947.
38. BROWN, J. B., and FRYER, M. P.: Management of compound facial injuries. Am. J. Surg. **75**: 625–630, Nov. 1948.
39. IVERSON, P. C.: Surgical removal of traumatic tattooes of face. Plast. & Reconstruct. Surg. **2**: 427–432, Sept. 1947.
40. PEACHER, W. G.: Speech disorders in World War II. Plast. & Reconstruct. Surg. **2**: 267–277, May 1947.
41. IVY, R. H.: Repair of facial defects by low-neck pedicle flaps. Plast. & Reconstruct. Surg. **1**: 119–123, Sept. 1946.
42. PEACHER, W. G.; CANNON, B.; and BROWN, J. B.: Repair of skull defects. Surgery **22**: 611–618, Oct. 1947.
43. CANNON, B.; BROWN, J. B.; and PEACHER, W. G.: Repair of scalp defects. Surgery **22**: 619–626, Oct. 1947.
44. KAZANJIAN, V. H., and WEBSTER, R. C.: Treatment of extensive losses of scalp. Plast. & Reconstruct. Surg. **1**: 360–385, Nov. 1946.
45. OWENS, N.: Reevaluation of implantation of fascial strips through masseter muscle for surgical correction of facial paralysis. Ann. Otol., Rhin & Laryng. **57**: 55–64, March 1948.

46. OWENS, N.: Implantation of fascial strips through masseter muscle for surgical correction of facial paralysis. Plast. & Reconstruct. Surg. **2**: 25–36, Jan. 1947.
47. BROWN, J. B., and MCDOWELL, F.: Support of the paralyzed face. J. A. M. A. **135**: 18–20, Sept. 6, 1947.
48. BROWN, J. B.; MCDOWELL, F.; and FEYER, M. P.: Facial paralysis supported with autogenous fascia lata. Ann. Surg. **127**: 858–862, May 1948.
49. ADAMS, W. M.: Multiple muscle substitution for correction of facial paralysis. Am. J. Surg. **74**: 654–664, Nov. 1947.
50. WHITE, J. C., and HAMM, W. G.: Primary closure of bedsores by plastic surgery. Ann. Surg. **124**: 1136–1147, Dec. 1946.
51. KOSTRUBALA, J. G., and GREELEY, P. W.: Problem of decubitus ulcers in paraplegics. Plast. & Reconstruct. Surg. **2**: 403–412, Sept. 1947.
52. BLOCK, M. A., and TSUZUKI, M.: Observations of burn scars sustained by atomic bomb survivors: a preliminary study. Am. J. Surg. **75**: 417–434, March 1948.
53. TRUSLER, H. M., and BAUER, T. B.: Keloids and hypertrophic scars. Arch. Surg. **57**: 539, 1948.
54. YOUNG, F., and BENJAMIN, J. A.: Repair of hypospadias with free inlay skin graft. Surg., Gynec. & Obst. **86**: 439–451, April 1948.
55. PRICE, R. A., and PENNA, O. J.: Effectiveness of drugs in preventing postoperative penile erections. Surgery **24**: 980–982, Dec. 1948.
56. DAVIS, A. D., and BERNER, R. E.: Primary repair of total avulsion of skin from penis and scrotum. Plast. & Reconstruct. Surg. **3**: 417–423, July 1948.
57. BYARS, L. T.: Surgical correction of nasal deformities. Surg., Gynec. & Obst. **84**: 65–77, Jan. 1947.
58. STEFFENSEN, W. H.: Reconstruction of nasal septum. Plast. & Reconstruct. Surg. **2**: 66–71, Jan. 1947.
59. MALINIAC, J. W.: Compressive suspension splint for severely comminuted fractures. Plast. & Reconstruct. Surg. **2**: 331–335, July 1947.
60. RUBIN, L. R.; ROBERTSON, G. W.; and SHAPIRO, R. N.: Polyethelene in reconstructive surgery. Plast. & Reconstruct. Surg. **3**: 586, 1948.
61. SAFIAN, J.: A critical review of recent literature on rhinoplasty. Plast. & Reconstruct. Surg. **2**: 463–473, Sept. 1947.
62. BLAIR, V. P., and ROBINSON, R. R.: Primary closure of harelip. Surg., Gynec. & Obst. **86**: 502–503, April 1948.
63. BROWN, J. B.; MCDOWELL, F.; and BYARS, L. T.: Double clefts of lip. Surg., Gynec. & Obst. **85**: 20–29, July 1947.
64. BROWN, J. B., and MCDOWELL, F.: Surgical repair of cleft lips. Arch. Surg. **56**: 750–757, June 1948.
65. FOGH-ANDERSEN, P.: Stein-Estlander-Abbe operation. Plast. & Reconstruct. Surg. **3**: 186–188, March 1948.
66. KAZANJIAN, V. H.: Estlander-Abbe operation in treating secondary harelip deformities and defects of upper lip resulting from cancer. Plast. & Reconstruct. Surg. **2**: 307–325, July 1947.
67. DORRANCE, G. M., and BRANSFIELD, J. W.: Studies in anatomy and repair of cleft palate. Surg., Gynec. & Obst. **84**: 878–885, April 15, 1947.

Temporary Lower Extremity Cineplasty

ROY C. ROUNDS, *Major, U. S. A. F. (MC)*[1]

THE Italians were the first to use cineplastic prostheses for lower extremity amputations. Although they have reported excellent functional results using quadriceps motor tunnels, the method has never been popular in this country. In selected patients with amputations of the upper extremity where delicacy of motion is more important than power of motion, the cineplastic prosthesis is commonly used. In the lower extremity, lack of any need for delicate motor function and the fact that modern artificial legs are so satisfactory mechanically, precludes the use of cineplastic devices.

The case presented here is unusual because a lower extremity cineplastic procedure without an amputation was performed as a means of preserving quadriceps muscle strength.

CASE REPORT

The patient was struck by shell fragments on 9 September 1944 in France. He sustained multiple wounds of the lower extremities, the right upper extremity, and the chest with severely comminuted fractures of the distal end of the right humerus and of the left femur. The lower third of the left quadriceps femoris muscle was completely avulsed. Infection appeared in the left femur, and in spite of repeated sequestrectomies and skin grafts, drainage persisted from several sinuses about the site of the fracture. Solid union of the femoral fragments occurred, however, 15 months after injury. A quadricepsplasty, which was an essential step in restoring function to the extremity, was repeatedly postponed because of the infection. In the meantime, wasting and contracture of the quadriceps muscle occurred. For this reason a quadriceps cineplastic procedure was performed well above the infected area. When healed, a molded leather cuff to grasp the calf of the leg was made. From the cuff a leather "quadriceps tendon" was attached to a plastic spindle passed through the motor tunnel. With this apparatus the patient was able to perform quadriceps setting exercises against gradually increased resistance and, in about 2 months, he had acquired sufficient strength to extend the leg momentarily against the force of gravity (fig. 1). He was also able to discard the brace he needed previously to stabilize his knee while walking (fig. 2).

[1] William Beaumont General Hospital, Fort Bliss, Tex.

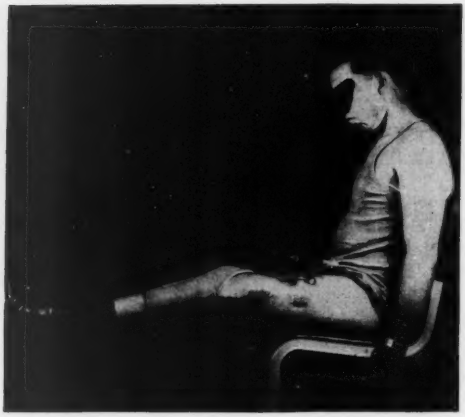


Figure 1.

Figure 2.

Ten months after the cineplasty and five months after complete cessation of drainage from the osteomyelitis sinuses in the lower thigh, the cineplasty was discontinued and a successful quadricepsplasty was performed. Drainage from the lower thigh did not recur, and on 16 March 1949, five months after the quadricepsplasty, he was discharged from the Army on a C. D. D.

Experiences With The Marrow Nail Operation According to the Principles of Kuentscher[1]

Tibia Fractures

Part VI[2]

PROF. DR. C. HAEBLER

The author is very reluctant to nail old gunshot tibia fractures, the wounds of which are healed, because of experiences in nailing old simple fractures. In such fractures (the sites of which were not located in the middle third where a stabile osteosynthesis could not positively be expected) he confined himself to a resection of the fibula, immobilization of the fracture, either directly or by means of wire extension, and a walking cast. Fixation was obtained by this method in from 8 to 12 weeks in five fractures that were from 9 to 14 months old.

The nailing operation and resection have been performed in only two cases where the fracture site was in the middle of the bone. These fractures were 10 and 11½ months old and the wounds had been healed for 3½ and 4 months. Both cases united without infection. Only the periosteum and soft parts were sutured, the skin left open, and a drain inserted as usual.

One of the patients could use his limb 3 weeks after the operation and 6 weeks later he was drafted for labor service. The joints were freely movable and the shortening amounted to 1 cm. The nail was removed 4 months after the operation.

---

[1] Translation prepared by the U. S. Naval Technical Unit, Europe (Medical Section), Office of Naval Advisor, Office of Military Government (U. S.) edited by U. S. NAVAL MEDICAL BULLETIN.

[2] Parts I to IV were published in the U. S. NAVAL MEDICAL BULLETIN beginning with the May–June 1949 issue. Part V was published in the January 1950 ARMED FORCES MEDICAL JOURNAL.

In the case of the second patient the final healing was delayed because the fibula was not resected (figs. 94*a* and 94*b*). The fracture

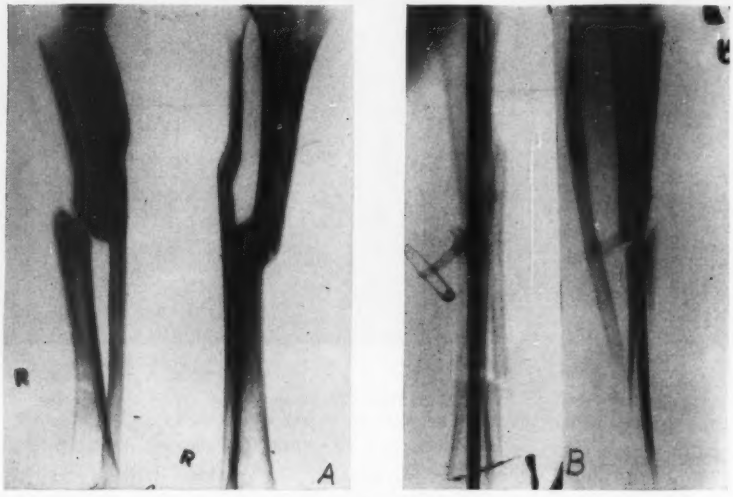


Figure 94 a and b.

united quickly and osteosynthesis was apparently stabile. During weight bearing the patient complained of pains at the fracture site. A roentgenogram (fig. 94*c*) showed marked rarefactions next to the

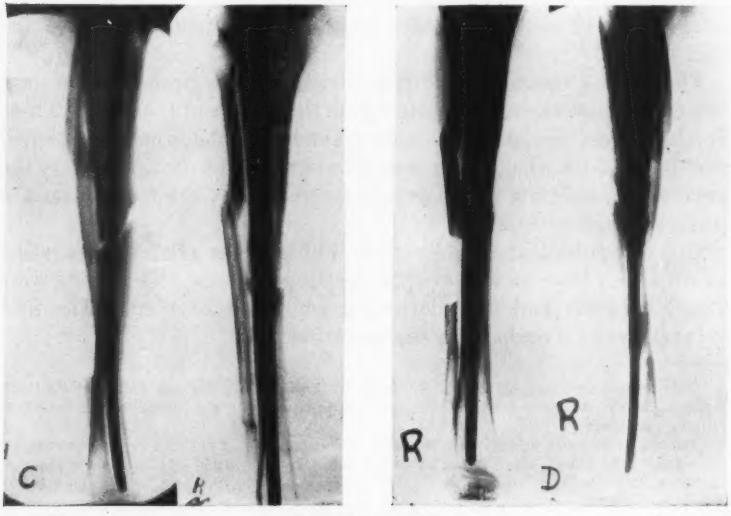


Figure 94 c and d.

nail point. The fibula was resected and the leg put in a U-shaped plaster splint and the patient began weight bearing. Eight weeks later the fracture had united (fig. 94*d*) and 4 weeks after that the nail was extracted.

If the fibula had been resected when the osteotomy was performed the fracture would have united much sooner.

Surgeons in military hospitals were too liberal with nailing operations. Out of seven nailed fractures of the tibia only three were suitable for nailing and only a relatively stabile osteosynthesis could have been expected in the others.

The operative site, in all cases, had been primarily sutured (some without drains), although the original wounds had been healed only 2 to 4 months. All these cases, with the exception of one with a stabile osteosynthesis, resulted in chronic suppurations, sequestra formation, and in one case (in which the wounds had not been opened) an osteomyelitis with abscess at the entrance site of the nail and suppuration of the prepatella bursa.

A stay in the hospital for from 10 to 16 months was necessary except in one case that united primarily; the results were decidedly poor and inferior to those which could have been obtained with conservative methods.

The officiousness and impatience of the surgeon is typical in the following two cases.

In a 15-month-old gunshot fracture (fig. 95*a*) the wounds of which had been healed for 2 months, the fibula was resected. The surgeon, however, failed to reduce and fix the fracture; diathermy was used, and when no callus formation was observed after 4 weeks (not abnormal under the circumstances) the fracture site was exposed, the fracture ends, which were closed by connective tissue, were freshened and united by only one nail which was far too short (fig. 95*b*). Drainage of the fracture cleft toward the calf, suture, and plaster cast followed. The wound had to be opened 8 days later at which time several small sequestra were ejected. The nail was removed 4 months after the operation although no callus was observed because the wounds were still infected. The fracture cleft was still visible (fig. 95*c*) and the fracture still movable. Three months later the fistula closed and after the leg was put in a walking cast for 2 months the fracture united. The final result was a shortening of 3 cm. and ankle and knee joints stiffened 50 percent.

In the preceding case as in the following case the nail was useless.

Figure 96*a* is a roentgenogram of a 13-month-old gunshot fracture, the wounds of which had been healed for 2 months. The same surgeon who performed the operation in the preceding case performed a fibula resection when the fracture was still movable but the leg was

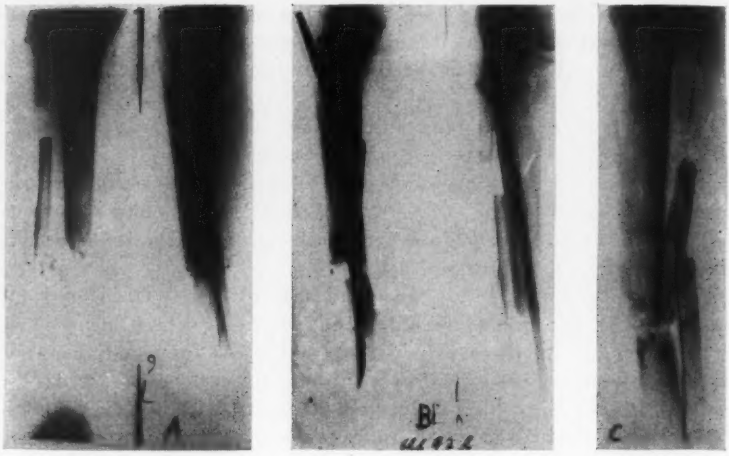


Figure 95.

not immobilized in a plaster cast. An open marrow nailing was performed 4 weeks later. On this occasion "part of the wall of the disrupted fragment had to be removed as being an obstacle to proper adaptation." By doing this the nail laid laterally in the proximal fragment and the fracture ends were pushed together causing a shortening of 4 cm. (fig. 96*b*). The sutured operative wounds became infected and several sequestra were ejected. The leg was put in a plaster cast and in doing this a serious infection of the marrow cavity was avoided. The distal fragment showed a distinct loosening of the bone structure. The nail was removed 4 months after the operation

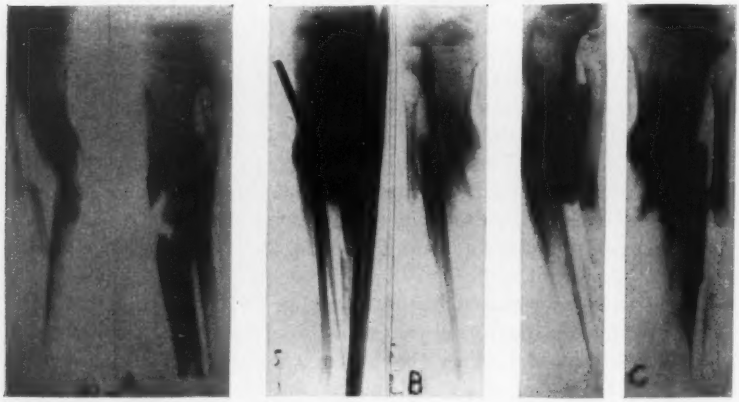


Figure 96.

because the wounds were still fistulous. Although the fracture was springy, a Volkmann splint was applied, the result being a slight antecurvation; the leg united in a valgus position with a shortening of 4 cm.; the further results were a stiff ankle joint, and the knee movable between 180° and 100° (fig. 96*c*). These impediments could have been avoided if the fracture had been properly reduced after resection of the fibula and the leg put in a walking cast.

In an effort to obtain a better fixation of the fracture and a quicker elimination of infection, tibia fractures were nailed without exposing the fracture site, but instead the sequestrum was removed by enlarging the fistula and a closed nailing was performed. A quicker reemployment of the patient has resulted in some instances but it must be admitted that similar results could have been obtained without the nail. Nailing was of no material advantage in fractures less than 3 months old. If conservative methods had been employed the time of treatment would have been shorter and the final result just as good. The author had only 3 out of 11 fractures where full mobility of the joints was obtained. Suppurations occurred, especially in cases in which approximation sutures were used.

It may be said that although these difficulties could have been encountered if conservative treatment had been used; these conditions are an ever-present danger in gunshot fractures.

Nailing does not always offer a clear-cut advantage even when the fracture is suitable for nailing and the osteosynthesis is perfectly stabile. This fact is illustrated by the following cases.

In a 10-day-old infected gunshot fracture (fig. 97), an attempt was made to obtain a better fixation and to control the suppuration by use of the marrow nail. During the operation not only the transversely located splinters were removed, but also those bridging over the medial side of the fracture, although they were connected with the periosteum. It was necessary then to push the fracture together after nailing, and this caused a shortening of 3 cm. Instead of the nail having a favorable influence on the suppuration, abscesses developed both at the entrance site of the nail and

Figure 97.

at the fracture site. Several sequestra developed at the fracture site so the nail had to be withdrawn although the fracture had not united. After 11 months in a plaster cast the fistula began to subside and the patient was finally discharged from the hospital with a stiff ankle joint and a 50 percent impediment of the knee joint.

If the slight antecurvation had been corrected and the leg put in a plaster cast at the beginning the shortening probably would have been less because the medial splinters would have presented a good matrix for the callus; the healing would have been quicker; and, in all probability, the mobility of the joints would have been better.

In another case a 4-week-old fracture (fig. 98*a*) was nailed because the lateral displacement could not be compensated for by extension fixation because the proximal fragment lay free of the granulating wound. A stabile osteosynthesis was expected and it was thought that early use of the limb would stimulate the apparent deficient callus formation. The fragments were reduced without special difficulty and without wide exposure by inserting the fingers into the wound. The nailing was uneventful. Figure 98*b* shows the fracture 6 weeks after nailing. The loose bone chips that were removed at the time of operation caused the slight defect at the front edge, but from a clinical point of view, the fracture was stabile. However, the nail was too short. The suppuration gradually subsided and 8 weeks after the operation the patient began exercises which gave him no pain. A dark secretion, which became almost black, appeared on the wound

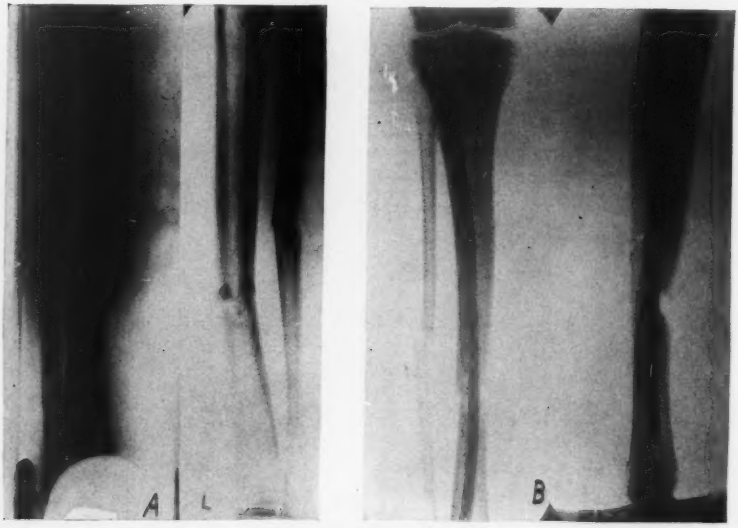


Figure 98 a and b.

and the fracture cleft showed a distinct loosening of the structure with the forming of sequestra, an indication of rust damage (fig. 98*c*). The nail was removed 3½ months after the operation although very little callus had formed. The rarefactions next to the point of the nail indicated that the nail had loosened and there was a thin black coating on the nail, a sign of corrosion. After the removal of the nail a small sequestrum was ejected and the wound healed. Since callus formation was still insufficient after 8 weeks in a plaster cast the united fibula was resected (fig. 98*d*). Six weeks after the resection of the fibula callus had increased and was more compact. From a clinical point of view the fracture had united (fig. 98*e*); a walking

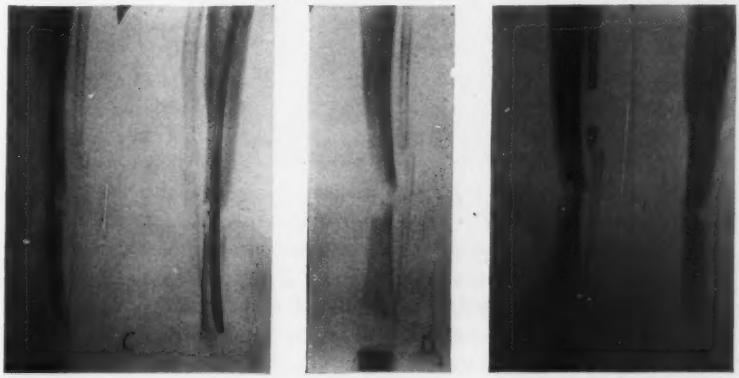


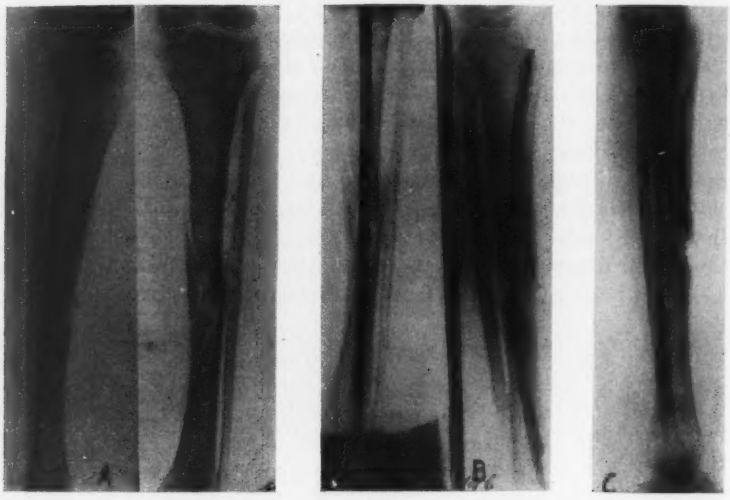
Figure 98 c, d, and e.

cast was applied. Nine and a half months after the operation there was a shortening of the limb of one-half cm., mobility of the ankle joint was slightly impeded, and the mobility of the knee was 180° to 90°.

In the case cited it must be admitted that the nailing was of no advantage, but, on the contrary, the necrosis at the fracture cleft would have been avoided if the nail had been omitted. Union was delayed because of the obstructing fibula and could not have been avoided by the use of the nail.

The same is true of other fistulous tibia fractures. The fact that a fracture heals with full junction 7 months after the operation is no special success in a 6-week-old oblique fracture from which a sequestrum was removed. On the other hand there is danger of the nail penetrating the soft parts unnoticed if a large fragment is disrupted from the proximal fragment. If this happens the nail would cause spreading of infection, abscesses, and chronic suppurations.

In the opinion of the author it is out of place to nail a tibia fracture such as in figure 99*a* for the reason that the fracture was still movable



Figue 99 a, b, and c.

4½ weeks after the accident; this is also true if a closed nailing is performed as is done in simple fractures. Figure 99*b* is the same fracture after nailing. Conditions were not aseptic and abscesses and suppurations followed, which necessitated additional plaster casts. The nail was extracted 5 months later.

Six months after the operation (1 month after the removal of the nail) the fracture had united but the wounds were still fistulous (fig. 99*c*). The patient was released from the hospital 10 months after the operation with an impediment of the ankle joint and the knee motion amounting to 80° to 180°. A better result would certainly have been obtained without nailing. Tibia fractures due to gunshot injury which are still movable may be satisfactorily reduced by conservative methods.

It must therefore be concluded that the marrow nail offers no advantages in chronically infected gunshot fractures of the tibia and one should abstain from nailing because of the many dangers, even though exposure is necessary because of sequestra formation.

If early union of the fibula hinders the proper reduction of the fragments or if it is the reason for insufficient callus formation the resection with subsequent application of plaster casts will fit the purpose and will not constitute a hazard.

Fistulous fractures of the tibia 5 to 18 months old were nailed in 4 instances. The fibula had united in all of them and if we examine the roentgenograms closely it will be seen that a final union could

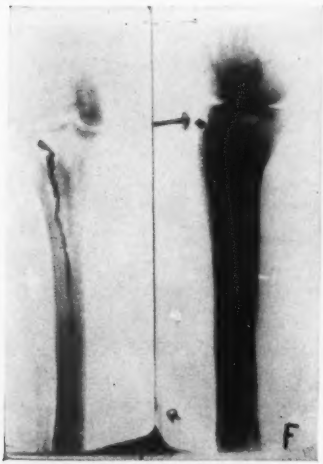
have been obtained by resection and proper reduction. This obvious solution was, as is so often the case, not attempted. In the case illustrated by figure 100 the author was not impressed by the fact that a pseudarthrosis had existed for 18 months, but was overenthusiastic about the marrow nailing method. In this case the nail was the cause of late complications which would very likely have been avoided had the nail not been used.

The patient wore a hinged splint, the knee joint was freely movable, but the ankle joint was slightly stiffened. After his discharge several sequestra were ejected, the last one 14 days before readmittance to the hospital. Figure 100*a* is a roentgenogram made on readmittance. After osteotomy of the fibula and mobilization of the fracture we were successful in nailing the fracture without exposure of the fracture site. The fracture was clinically solid but there was a slight gap at the fracture cleft (fig. 100*b*). A small abscess developed at the entrance site of the nail (which was left open) 10 days after the operation. Except for this abscess, healing was uneventful and the patient began using the limb 3 weeks after the operation; the fistula had then closed. Weight bearing caused the gap at the fracture cleft to close (fig. 100*c*) and a small sequestrum was ejected. The wounds closed and the patient was then treated as an out-patient 2½ months after the operation. The fistula broke open sometime later and secreted a dark fluid. After 5 months union was sufficient to permit the withdrawal of the nails; the outer nail had rusted on both edges so there remained only a 3 mm. bridge at the fracture cleft, and the inner nail also had rusted. The nail had obviously obtained a firm hold in the

Figure 100 a, b, and c.

Figure 100 d and e.

callus that closed the marrow cavities in spite of the unfavorable location of the fracture; there were no rarefactions noticed at the point of the nail. A sequestrum had formed through the fracture cleft (fig. 100*d*). This sequestrum was removed after the entrance



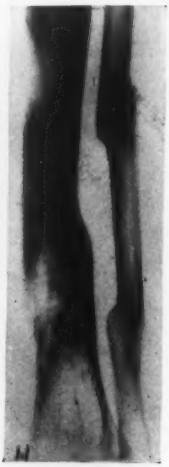


Figure 100 f, g, and h.

wound of the nail had healed by flattening of the bone. Six months after the operation the wound had healed and the patient was discharged from the hospital (fig. 100*e*).

Sometime after his discharge from the hospital the patient had pain and swelling at the fracture site which subsided when one of the wounds broke open. No sequestra were ejected. Upon readmission to the hospital 20 months after the operation no sequestra could be detected, but a contrast medium, introduced by approach from the tuberosity of the tibia revealed a fistula in the marrow cavity that reached to the middle of the tibia (fig. 100*f*). The fistula could not be traced as far as the fracture cleft at the time of operation, and a methylene blue solution injected through a urethral catheter did not appear at the fracture cleft (fig. 100*g*). The bone was then chiseled at the upper section of the canal and the fistula exposed (fig. 100*h*). The patient was discharged from the hospital 6 weeks after the last operation with wound healed.

There is no doubt that this long stay in the hospital would have been avoided if the nail had not been used.

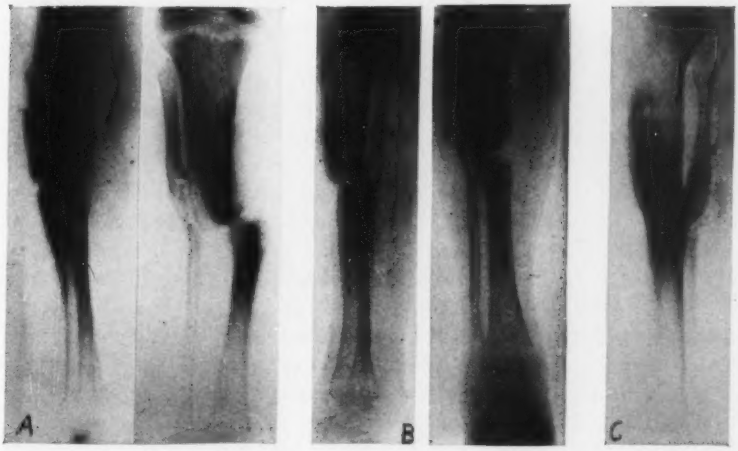


Figure 101.

In a 7-month-old fracture (fig. 101*a*) in which the roentgenogram suggested the beginning of a pseudarthrosis, we believe that a simple resection would have been better and would have led to quicker union. Connective scar tissue between the fracture ends which also closed the marrow cavities was found at time of operation. The fibula was not resected and when the nails were removed 4 months after the operation because of a dark-colored discharge which indicated rust, the fracture had not united (fig. 101*b*) (the nails had oxidized at the

fracture site). The wounds healed soon after the nails were removed and 4 months later the fracture had united sufficiently to allow the replacement of the walking cast by a Unna bandage (fig. 101*c*).

The author maintains that these fractures would have united satisfactorily if the fibula had been resected and a nonoperative reduction performed followed by subsequent application of walking casts, because they were not pseudarthroses but ununited fractures. To prove this contention attention is directed to figure 102*a* which is similar to the case illustrated by figure 101 in which the diagnosis of a pseudarthrosis seems fully justified. This "pseudarthrosis" united after the resection of the fibula and 6 weeks in a plaster cast (fig. 102*b*). The patient was able to perform his duties 8 weeks after resection. This is one example out of many such cases treated by the author. We have always been successful except in cases having serious bone defects.

Figure 102.

The nailing of old gunshot fractures of the tibia that are in bad position and the wounds just healed should be done only if it is a transverse or oblique fracture in the middle third and a stabile osteosynthesis is certain. Even if these conditions prevail great care should be exercised and it is recommended that nailing be done only when satisfactory position cannot be obtained by nonoperative methods after the resection of the fibula. The wounds must be drained and the

skin should not be sutured. An additional plaster cast will be necessary until danger of infection has passed.

In all other tibial gunshot fractures nailing should not be performed. They will become solid as a rule by placing the limb in a cast after the fragments have been brought to proper abutment.

If the fracture fails to unite one of the time-honored pseudarthrosis operations should be performed even though a pseudarthrosis exists or is imminent, if at least 1 year has elapsed after the healing of the wounds.

The nailing of old fistulous tibia gunshot fractures should not be performed even though a pseudarthrosis exists or is imminent. Nailing has no advantages and it constitutes an additional hazard of complications. The double nail is particularly susceptible to corrosion by suppuration and this type of nail further sustains the suppuration and is obviously the cause of sequestra.

GUNSHOT FRACTURES OF THE HUMERUS

Boehler advises against marrow nailing of pseudarthroses of the humerus because it has no advantage over the other methods, especially with respect to callus formation, and because it failed in case of atrophic bone and because of the danger of spreading infection throughout the entire marrow cavity.

In 47 cases of marrow nailing of old fractures of the humerus due to gunshot injuries an osteomyelitis occurred in 3 cases: a 3-month-old fistulous fracture; a 19-month-old pseudarthrosis, the wounds of which had been healed for 5 months; and in a 2½-year-old pseudarthrosis that had been operated on 13 months after the wounds were healed. The spreading of infection, doubtless, was caused by improper technique and failure to open the wounds soon enough. The spreading of infection into the marrow cavity can be avoided by immediately opening the wounds wide as soon as an infection develops, even if the osteosynthesis is not completely stabile.

A primary closure of wounds is always dangerous in old gunshot fractures because one is never on the safe side regarding infections even if the fistulae are closed for more than a year. Only four out of eight of that type of pseudarthroses united primarily. On the other hand, three out of five fractures which had been nailed 3 to 5 months after the closure of the fistulae and sutured primarily with adequate drainage united without infection. Postponing the operation apparently had no advantage.

The suppuration was, if no osteomyelitis occurred, chronic in all cases where the wounds had not been widely opened and where only a simple drain had been used. In contrast to this six pseudarthroses, which had been operated upon 4 to 6 weeks after the wounds had

healed, united by bone granulation much quicker because the skin was not sutured and the wounds were thoroughly drained; the musculature over the bone was held in place by a few stitches.

There were no sequestra observed in the 11 cases in which the wounds had healed less than 5 months prior to the operation, although there were a few compound fractures among these cases.

Fourteen pseudarthroses from 5 to 22 months old were nailed by the author regardless of the existing fistulae. The wounds were thoroughly drained and left open, therefore, not one case of osteomyelitis developed and sequestrum formation was observed in two cases, although the osteosynthesis was not absolutely stabile in all of the cases and existing sequestra (which was removed at the time of operation) was the cause of the suppuration.

The author feels justified in denying the possibility of infection spreading into the marrow cavity when the nail is used. This danger is completely avoidable and should not be considered a contra-indication to nailing pseudarthroses of the humerus. All 33 of our cases of pseudarthrosis following gunshot injury resulted in bony union. (One case, an 8-month-old pseudarthrosis in the distal third, had a bone graft operation performed 5 months after the operation with the nail in position, by a very "operation happy" surgeon. It is doubtful whether this was necessary.)

The treatment in the hospital was prolonged and lasted, in many cases, more than a year, the nails were not removed for from 6 to 7 months. The desire to create better conditions, that is, to get better joint mobility by adequate movement, was largely responsible for the long hospitalization. The nail was left in position in these cases as an additional fixation and the patients so treated were farmed out for work, so the time spent in the hospital cannot be considered as a measure of unemployability.

The fact that all of the pseudarthroses resulted in a bony union, including some cases of severe bone atrophy and cases in which a bone graft had previously been performed without success, refutes the opinion of Boehler. In a majority of these cases the osteosynthesis was stabile, at least to such an extent that the nail prevented any lateral movement. If the fracture was movable longitudinally, which happened in several cases, the fracture was pushed together and an abduction plaster cast applied. The arm was elevated above the horizontal until the fracture united. If the nail became loose, especially when it was introduced by approach from the distal side, no additional hazard was presented if treated properly. The advisability of using a longitudinal wire suture to prevent distraction is uncertain because if the bone is atrophic the wire is liable to cut through the bone and

prove dangerous instead of helpful. This is illustrated by the following examples:

In a 6-month-old gunshot fracture (fig. 103*a*), the wounds of which had been healed for 6 weeks, the broken-out bone wedge was not joined to the distal fragment. The fracture was mobilized under anesthesia, although it appeared freely movable, as it was hoped to perform a closed nailing operation. A closed nailing was not possible because the Kirschner wire used as a guide repeatedly slipped off the proximal fragment so an open nail operation was performed 8 days later. On exposure it was found that the marrow cavity was closed by firm callus and that a pseudarthrosis existed between the broken-out wedge and the distal fragment. The connective tissue between the fracture ends was removed and the proximal marrow cavity was opened with an awl. The bone was not resected but the wounds were properly drained and the musculature at the fracture site, but not the skin, was brought into position by a few approximation stitches. The arm was then placed in an abduction cast; the osteosynthesis was clinically stabile (fig. 103*b*). As no fever occurred the drains were removed 10 days later and the arm subjected to movement exercises 3 weeks postoperatively. Six weeks after the operation the wounds had

Figure 103 a and b.

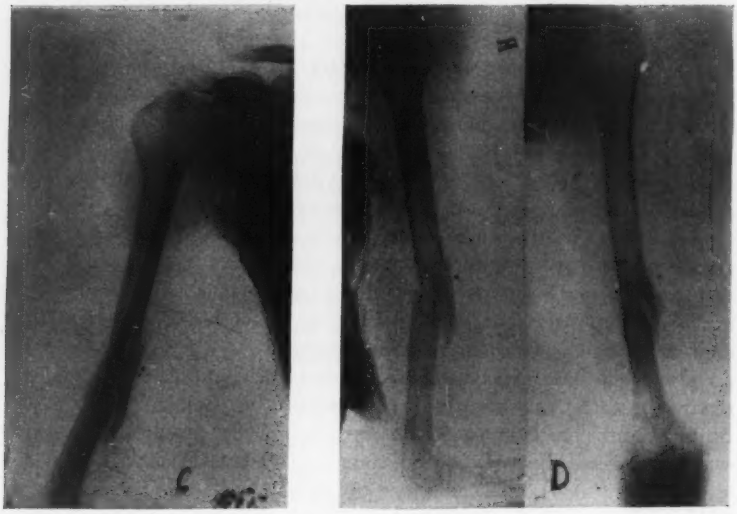


Figure 103 c and d.

healed secondarily so the plaster cast was removed. The slight gap of the fracture had been pushed together by use of the limb (fig. 103*c*) and 6 months after the operation the fracture had united (with a slight angulation) (fig. 103*d*) so the nail could be withdrawn. The patient had been detailed to work during the movement exercise period and when he was discharged to the troops there was a limitation of extension of the elbow of 25°, but the shoulder was freely movable. The elbow joint was nearly stiff when admitted to the hospital.

In another case the stay in the hospital was more eventful. This case was a 2-year-old pseudarthrosis (fig. 104*a*) where a bone graft operation had been performed 9 months after the injury but had been unsuccessful. Because the wounds had been healed for more than a year, the wound at the fracture site was sutured and drained. The wounds were opened 3 days later because the patient had fever. Although no osteomyelitis or sequestra formation occurred, the patient had pain and fever whenever exercises were begun, so the plaster cast was left in position. As the wound at the fracture site continued to suppurate, 7 months after the operation it was decided to remove the nail although the roentgenogram showed insufficient callus formation (fig. 104*c*). The arm was left in the plaster cast 6 weeks longer and the callus solidified to such extent that exercises could be started (fig. 104*d*). Eleven months after the operation the fistulae were entirely closed and the patient was discharged, but the mobility

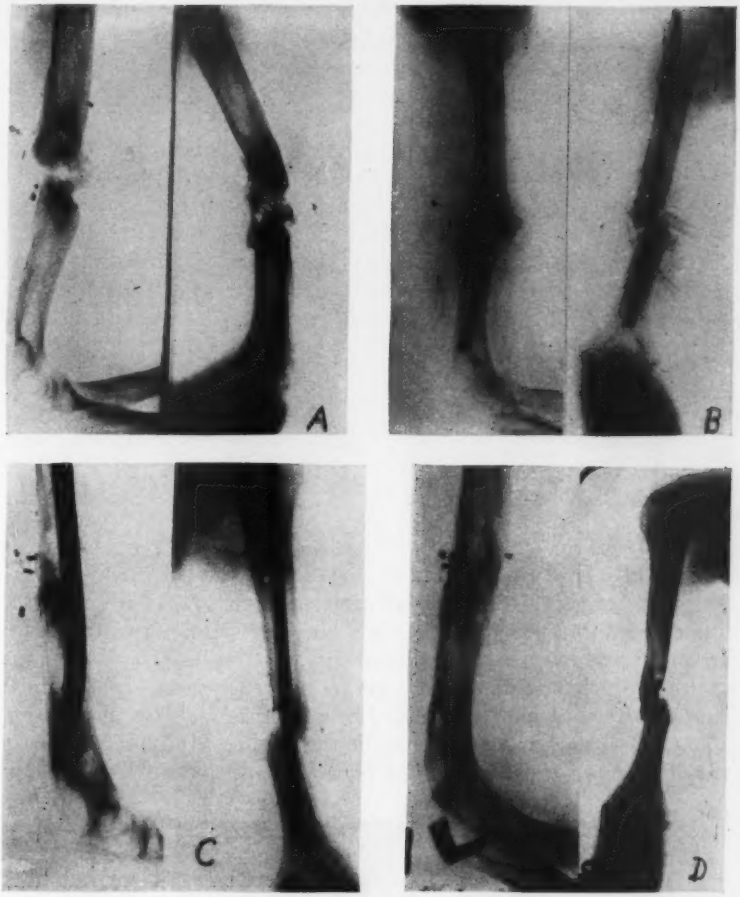


Figure 104.

of the joints was not notably improved, which was to be expected in a 2-year-old fracture.

No reliance can be placed on the solidity of small callus bridges. This was demonstrated in the nailing of an 8-month-old fistulous pseudarthrosis (fig. 105*a*) the outer nail of which had turned 90° so the inner nail could not be used. The fracture appeared solid (fig. 105*b*). Eight days later the fracture could be moved longitudinally and was somewhat distracted (fig. 105*c*). After 9 months in an abduction cast the fracture became so solid by the formation of a callus bridge that the nail was removed (fig. 105*d*). The wound had

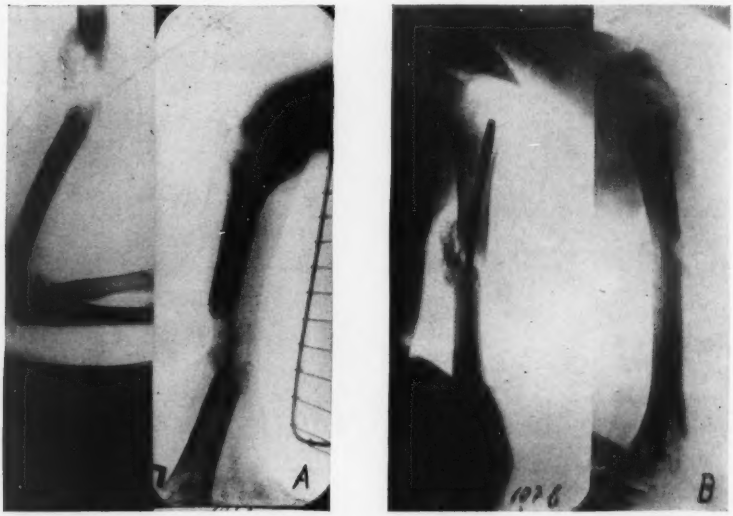


Figure 105 a and b.

healed but refracture occurred 2 months after the nail was removed. Since the fracture had not healed after 10 months in a plaster cast, it was decided to renail the fracture. The tapering point of the distal fragment was resected, the callus bridge was freshened, and after the nailing, which was done this time by approach from the proximal side, the fracture ends were united by a longitudinal wire suture (fig. 105*f*). The nail appeared short, but it was so firmly embedded in the distal marrow cavity that the fracture separated when an attempt

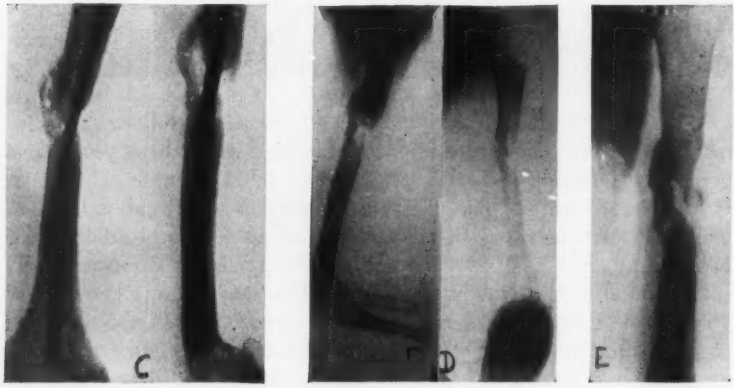


Figure 105 c, d, and e.

was made to drive it further. The wounds healed, the plaster cast was removed 12 weeks later, and the nail withdrawn 6 months postoperatively. A fistula occurred at the fracture site because of the wire suture; this was then removed. Nine months after the second operation, and 2½ years after the first operation, the patient was released from the hospital (fig. 105*g*).

A better result may have been obtained had the fracture been resected at the beginning, but as the pseudarthrosis was fistulous it was deemed advisable not to resect the fracture in order to preserve as much of the granulation tissue as possible and so prevent a medullary infection. If the osteosynthesis is very stabile these pseudarthroses will become solid although the fracture ends do not widely abut but are only bridged over by callus splinters, as was demonstrated in the following case.

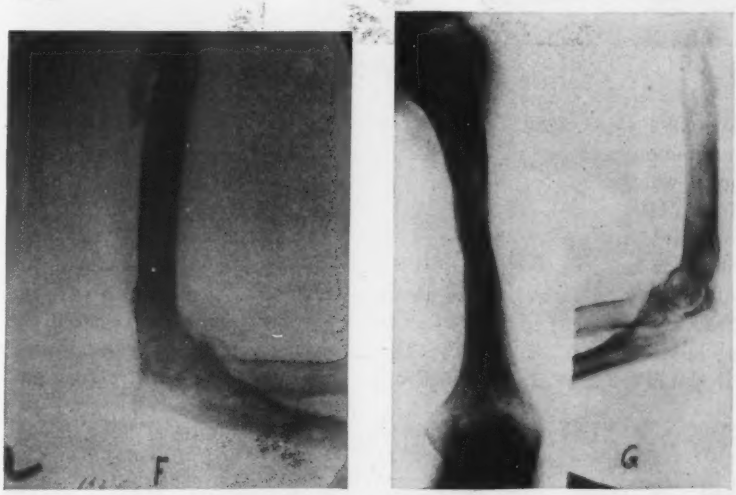


Figure 105 f and g.

In a 6-month-old fistulous ununited gunshot fracture of the humerus (fig. 106*a*) two small sequestra were removed at the time of nailing. The bone splinters, which were connected to the periosteum and partially connected to each other by callus, were left in position although they made it impossible to push the fracture together. The nail, which was driven in as far as the head, made the fracture solid although the nail was free of the defect (fig. 106*b*). The musculature over the exposed nail was held in place by an approximation suture and the wounds were properly drained and left open. The wound at the olecranon healed but a small sequestrum was ejected at the fracture site after which the fistula closed. Ten weeks after the operation

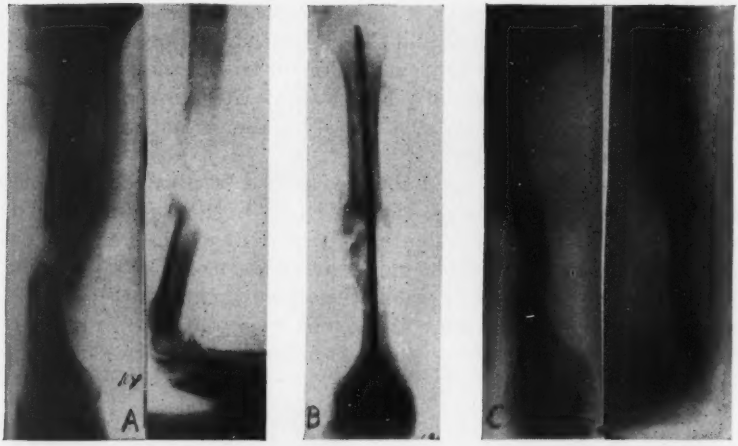


Figure 106.

exercises were begun, and a year postoperatively the fracture was solid enough to permit removal of the nail (fig. 106*c*). The shoulder joint was freely movable, but the elbow joint had a mobility of only 70° to 160°.

Pseudarthroses of the middle third of the humerus are very suitable for nailing even if the distal fragment is short. An example of this was a 22-month-old fistulous pseudarthrosis (fig. 107*a*), the fracture of which had united so much 5 months after nailing that the abduction cast was removed. The nail was visible in the open wound at the elbow and acted as a drain. The wound at the fracture site

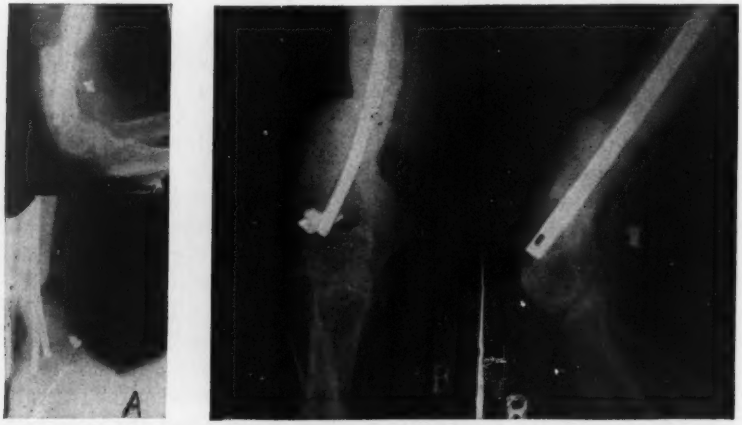


Figure 107.

healed quickly. The wound at the elbow healed, but the nail was not withdrawn because of insufficient callus formation. The patient was drafted for labor service and was not seen again for 2¾ years when he returned to the hospital because of an abscess on the elbow (fig. 107*b*). Since the fracture was united by bony union, the nail was removed when the abscess was incised. The elbow joint was movable between 90° and 160°. This certainly may be considered a good result.

In this type of fracture the nail must be introduced exactly at the upper edge of the olecranon fossa and the end of the nail bent slightly to prevent the distal fragment from slipping off the nail. If these points are observed it will be unnecessary to use a spiral spring or other special measures. If the nail is not introduced exactly in the middle the bone may be split and the osteosynthesis will not be stabile.

Figure 108.

It is recommended that gunshot fractures of the humerus, especially if they are fistulous, be nailed by approach from the distal side because of the ever-present danger of infection. By a distal approach the entrance spot of the nail will be the deepest point and the nail will form a perfect drain if the wound is left open.

This will not be possible in cases of fractures in the proximal third because of the danger of the nail slipping out of the head causing a pseudarthrosis. This actually happened in the case illustrated by figure 108 which had been nailed immediately after the injury. The case came to the author for treatment a year later. A union was secured by means of a staircase-shaped freshening and wire suture.

The authors and many others have attempted to nail such ununited fractures from the proximal side but complications have developed in every case.

One pseudarthrosis, the wounds of which had been healed for 2 months, became infected and resulted in suppurations with perforations toward the olecranon fossa; another, the wounds of which had been healed for a year, had an infection beneath the deltoid muscle; and in two other fistulous pseudarthroses the distal atrophic fragment was split, one having an infection of the elbow joint.

Although all four came to a bony union, the nail proved to be of no advantage. The more simple method and one more likely to be successful (especially if the bone is atrophic) is to point the distal fragment slightly and push it into the spongiosa of the head and then apply an abduction cast. This may be done in a fistulous fracture in which case no wire suture should be used because it will cut the atrophic bone and cause the resorption of the fracture ends and result in faulty union.

The advantage of nailing old fractures of the humerus in which an uneventful healing is not assured is that it can be performed while the wounds are suppurating. We have nailed 14 fractures of this kind. Sequestra formation is expected in this type of fracture especially if compound, but it would occur if conservative methods were used. Osteomyelitis developed in one instance and this could have been avoided.

In dealing with old fractures it is important that only sequestra be removed, the splinters should be left in position and the fracture not be freshened; the removal of the splinters destroys the protective granulation dam. It is also important that the osteosynthesis be stabile, that the wounds be properly drained, and that neither the entrance site of the nail nor the wounds be sutured to avoid infection.

The nailing should be performed, if possible, by approach from the distal side to avoid infection of the marrow cavity. The nail will act as a drain and its entrance site will be the lowest point. If it is necessary to nail from the proximal side it will be necessary to apply an abduction cast and to elevate the arm above horizontal until danger of infection has passed.

The following case describes the conditions that may arise in case of an atrophic bone which must be given special consideration.

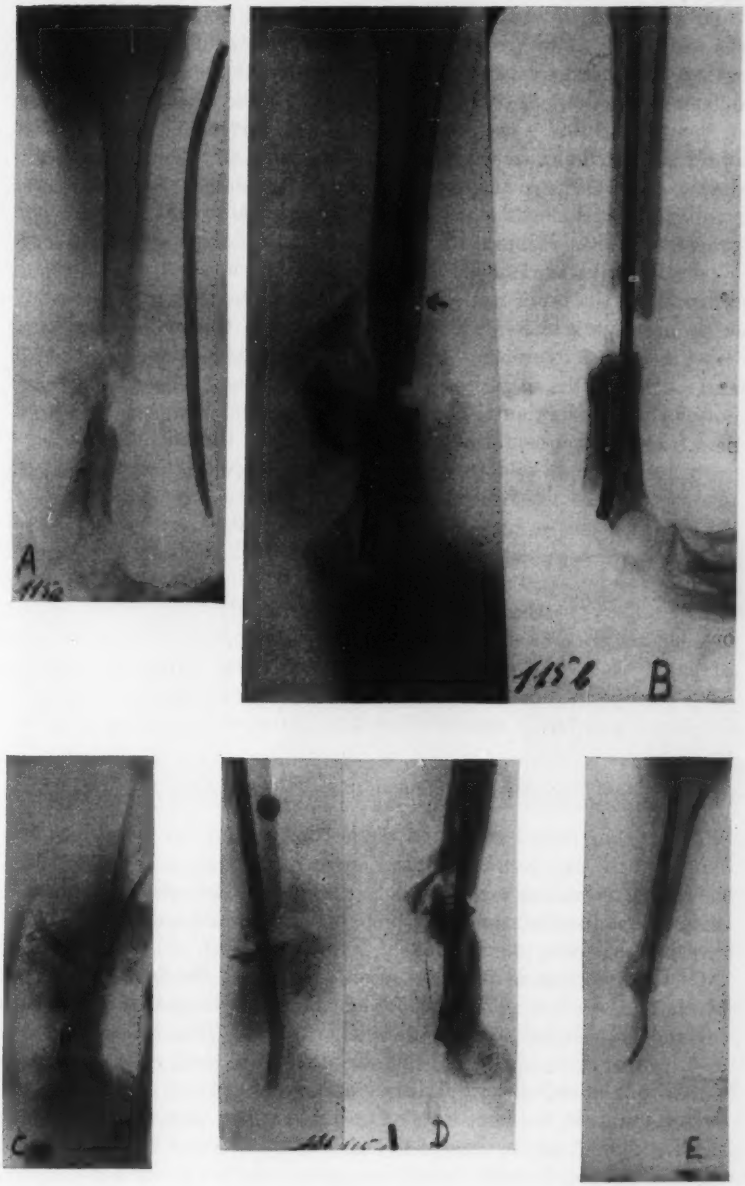


Figure 109.

In nailing a 16-week-old fistulous compound fracture (fig. 109*a*), the nail stuck in the cortex when driven into the proximal marrow cavity. The nail was withdrawn and the point bent slightly and was driven in again. The fracture being movable after the nail was driven in (fig. 109*b*), the inner nail was also driven in. The fracture could be distracted so it was pushed together and placed in an abduction cast. The roentgenogram taken after the abduction cast was applied showed the fracture to be distracted and that the nail had penetrated through the cortex into the soft parts (figs. 109*b* and 109*c*). When the nail was removed the point of which was palpable through an incision, the outer nail slipped out of the distal fragment, so another nail 2 cm. longer was used. The end of this nail was shaped like the letter **S** to assure a firm hold in the proximal fragment. This nail reached to the spongiosa of the head. A piece of bone had broken out at the flexor side of the distal fragment and had to be removed. The fracture then could be easily adapted, but the nail did not have a proper hold and had to be fixed by wire loops. The position was satisfactory but the fracture still could be distracted (fig. 109*d*), so considerable pressure was exercised from the side of the elbow and an abduction cast applied. The wounds which had been left open healed and the fracture had solidified to such an extent that movement exercises could be started 4 months postoperatively (fig. 109*e*). Four weeks later the patient was transferred to a special hospital because of paralysis of the radial nerve. The nail was removed 8 months after the operation and after the nerve had been sutured. The shoulder joint was freely movable but the elbow was movable only 90° to 170°.

PSEUDARTHROSES OF THE FOREARM

The author nailed no old fistulous fractures of the forearm. The danger of the nail penetrating the cortex of the bone is great although the course of healing is aseptic. If a pseudarthrosis occurs or already exists large areas of bone may become necrotic and a defect pseudarthrosis may ensue.

Of the five cases of pseudarthrosis of bones of the forearm treated by the author, the wounds of which had been healed from 4 weeks to 9 months, all but one came to a bony union. This failure was due to improper technique. In this case we had to deal with a pseudarthrosis of the radius with fibrous cartilage (fig. 110*a*). As the ulnar defect was 3 cm. the bone ends of the radius were resected to the same extent. The fracture ends of the ulna did not have a wide abutment; the freshened ends were about 1 cm. apart, which was considered good enough. When the nails were driven in there was still a gap in the ulna. To avoid further resection a wire loop was used to bring the

bone ends together and to prevent longitudinal displacement a longitudinal wire suture was also used (fig. 110*b*). A wound infection developed at the ulna and the point of the distal fragment sequestered resulting in an ununited fracture of the ulna, but the radius united (fig. 110*c*). Shortly after the sequestrum was ejected and the wire removed the fistula closed. Five months postoperatively the nail was withdrawn and a month later the patient was discharged from the hospital. Since the function of the limb was not seriously hampered by the ununited fracture of the ulna nothing was done to correct the condition.

In the preceding case the nail was too short and did not hold in the marrow cavity. The point of the nail penetrated the cortex. The author warns against the use of a wire loop. An angulation cannot be avoided by its use if the osteosynthesis is not stabile; the bone will slip out of the loop, and if an infection develops resorption

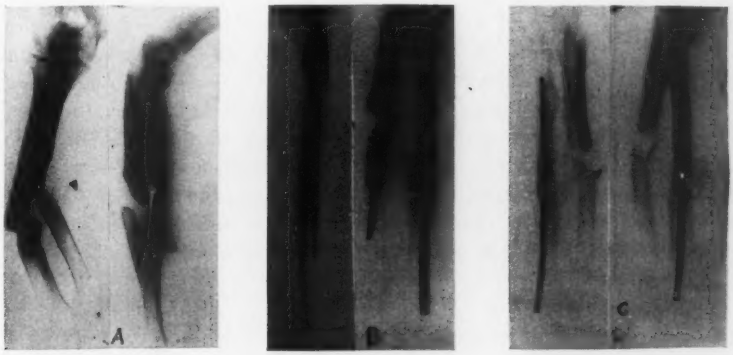


Figure 110.

in the area of the wire and formation of sequestra is imminent, especially if the bone becomes atrophic. The longitudinal wire is justified as it avoids distraction, but it should be applied as widely as possible. If the bone is atrophic this wire is dangerous.

If excessive resection is to be avoided the best method to be used is to nail the radius, apply a plaster cast, and wait until the radius unites, then perform a bone graft of the ulna.

It is useless to perform an osteosynthesis and reduce the head of the radius by nonoperative procedure in cases of Monteggia fractures. If the marrow nail is used the head of the radius will slip off and the ulnar nail will break or perforate the bone. The radius luxation must be reduced by operation and the ulna nailed at the same time. A union can be achieved by this method. A Lane's plate

will not be sufficient, especially if it is fixed with two screws as shown in figure 111*a*. This was a 12-month-old pseudarthrosis of the ulna with a central luxation of the radius. The wounds were healed 2 months after the injury and 2 months later an operative reduction of the radius and fixation of the ulna by a Lane's plate was performed. As there was but one screw on each side of the plate it did not hold and another luxation of the ulna and a pseudarthrosis developed. The cartilage of the radius head had been entirely destroyed because an extensive resection of the radius head had been made in a previous operation. The connective tissue callosities of the ulna were removed and a marrow nail inserted. The nail jammed in the callus of the distal fragment and had to be cut off (fig. 111*b*). Although the nail was too short the osteosynthesis was stabile and the wounds healed. A plaster splint was applied for 4 weeks after which exercise of the limb was started. Six months after the operation the fracture had united (fig. 111*c*) and no rarefactions around the tip of the nail were noted. Rotation of the forearm was reduced one-fourth but all other joints were freely movable.

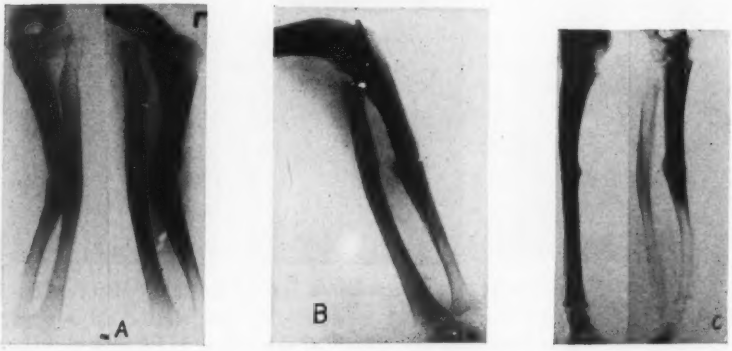


Figure 111.

If the head of the radius is resected too sparingly the longitudinal wire will be of no benefit and the result will be either a distraction and faulty union or a fracture of the nail.

It is the author's opinion that the ulna should not be resected or both bones be nailed after resecting in cases of Monteggia fracture. If the resection is done sparingly a new luxation is likely to occur and if infection develops, especially if both bones are nailed, the outcome will be worse than before the operation. Most of these fractures will unite because the obstruction of the ulna is offset by the distal luxation.

If the fracture of the radius has united but the function of the wrist is seriously hampered by a luxation of the ulna in the distal radio-ulna joint, a resection of the ulna in the shaft and nailing will be useless. The shrinking of the scar at the wrist joint will cause a new distraction of the site of resection and a new pseudarthrosis will develop in spite of the plaster cast. It would be better to resect the styloid process of the ulna.

Two cases of defect pseudarthrosis of the ulna, in which a bone graft had been made, were nailed with the idea that the nail would aid in uniting the graft by holding it in position. (This procedure has been recommended by others but Boehler refutes it because he claims that the graft does not get enough nutrition because of the destruction of the cavity.) Infection developed in both of these cases and a faulty union resulted, although the osteosynthesis was stabile in both.

Because the author saw two cases of tibia pseudarthrosis that resulted in faulty union he refrained from further experiments. The plaster cast assures adequate position of the forearm if left on for about 12 weeks. The idea that the marrow nail might interfere with nutrition of the bone is by no means farfetched. As a matter of fact zones of resorption are usually seen at the wire loops when a graft is used. The patient material of the author was not large enough to draw definite conclusions as to the merits of using the marrow nail, and as we have had good results with the bone graft without it, we are inclined to advise against using the nail when a bone graft is made as any advantage would be offset by danger of infection.

SUMMARY

The marrow nail is advantageous in pseudarthroses due to gunshot fracture of both bones of the forearm if a stabile osteosynthesis can be assured. It is necessary to resect wide enough to assure a wide abutment of the bones.

An attempt to achieve this abutment by the addition of a wire loop is dangerous. In case of infection this may cause a gradual fracture and formation of sequestra at the bone ends, especially if the osteosynthesis is not stabile and the bone is atrophic.

Only a longitudinal suture is necessary if the calcareous content of the bone is satisfactory and this should be applied widely. If the osteosynthesis is stabile it can be dispensed with.

If there is a defect pseudarthrosis of one bone, a bone graft is the most satisfactory treatment. An additional marrow nail might interfere with the nutrition of the graft. Resection of the obstructing bone and then nailing both bones is the least desirable method.

If there is an imminent pseudarthrosis following a Monteggia fracture it will be necessary to nail the ulna and reduce the luxation of the head of the radius by operation to avoid a faulty union or a fracture of the nail.

If a luxation has existed for more than 6 months it will be necessary to resect the head of the radius and nail the ulna pseudarthrosis. The resection must be wide enough to prevent the radius from becoming obstructive again.

In case of an old luxation in the distal radio-ulna joint the resection of the styloid process will give better results than the resection of the shaft of the ulna and then nailing.

Psychologic Approach to Hard of Hearing Patients

CLARE B. CORNELL, *Ph. D.*[1]

PSYCHOMETRIC tests show that patients with a loss of hearing score high on depression and hysteria and often show tendencies toward hypochondriasis and psychasthenia. They live in a partial or complete auditory vacuum that causes feelings of insecurity and suspicion. The sounds of a normal environment are missing. They misunderstand commands and are accused of being unintelligent. They fail to hear the sound of an approaching automobile until it whizzes past. When they enter a room and see a conversation in progress, they imagine that people are talking about them, because they cannot hear.

CASE REPORT

A 35-year-old man with over 5 years of service, was acting as manager of a hotel operated by the Army of Occupation and accommodating 500 women, civilian and military. He was happy in his job and popular with guests and military personnel until he received a reprimand from his commanding officer that he was neglecting his duties. About the same time a close friend, who was visiting him one evening, walked out without saying a word. The patient telephoned asking for an explanation. The friend replied, "I sat there while you were working on your books and asked you question after question, but got no reply. If you are going high hat, I'm through with you." It suddenly dawned on him that his hearing was failing. After a check-up, he was transferred to the Audiology and Speech Correction Center at Walter Reed General Hospital.

He appeared extremely depressed, sensitive, and rebellious on arrival at the Center. Psychometric examination revealed rather severe depression and hysteria. The otologic diagnosis was moderate deafness, perceptive type, cause undetermined. An audiometric examination showed a hearing loss for speech of 80 decibels in the right ear, and 40 decibels in the left ear, or a loss of 60 to 65 percent in the right ear and 30 to 35 percent in the left ear. The patient was fitted with an air conduction 22.5 volt hearing aid to the left ear, which afforded normal hearing. He received 4 weeks' auditory training, 5 weeks of lip reading, and 3 weeks of speech correction. The therapeutic results of this program were soon reflected in the personality of the patient. He regained self-confidence and became communicative, friendly, and alert. After 2 months he was given a C. D. D., because of bilateral deafness.

---

[1] The Audiology and Speech Correction Center, Walter Reed Hospital, Washington, D. C.

This case history has been outlined in some detail in order to emphasize the point that a person with a hearing loss, sufficiently severe to interfere with his daily activities, requires sympathetic understanding and skilled medical attention. This is equally true of patients in whom the cause is undetermined and of those with observable trauma received in combat. Whether the hearing loss is sudden or long standing, the net result is an insidious effect on the personality of the patient. There is a certain degree of psychogenic overlay in at least 90 percent of these patients. It is best to treat the patient as having a genuine hearing loss until positive evidence to the contrary has been discovered, even in the case of a suspected malingerer.

Harsh treatment by the doctor only aggravates the condition. The author recently saw a 23-year-old patient weep hysterically in an initial interview. He was asked to explain the outburst, after he had recovered his composure. He replied, "I couldn't help it. I've been kicked around so long that when someone treats me like a human being I just can't take it." His loss of hearing was not severe but, because of unskilled treatment, he had developed an anxiety neurosis resulting in overt hysteria. It is best to give the patient all possible information concerning his deafness, but only when such information is based on a careful examination. Another patient was told that he would eventually lose his hearing completely. The etiology and prognosis of his case did not justify such a statement, but the result was a distressing, although transitory, anxiety.

CONCLUSIONS

Medical officers should realize that loss of hearing often has serious psychologic complications. Readjustment to the hearing loss involves not only medical and surgical treatment or both, plus furnishing a mechanical aid when necessary, but also careful psychotherapy carried through from the first consultation until the patient is discharged from the hospital and returned to duty or to civil life.

Amicrobic Pyuria

SPENCER JOHNSON, *Captain (MC) U. S. N.*[1]
HENRY E. WOLFE, JR., *Lieutenant, junior grade (MC) U. S. N.*[1]

AMICROBIC pyuria is a disease entity which has been presented in the medical literature for the past two decades under such titles as: sterile pyuria; abacterial pyuria; true infective amicrobic pyuria; and amicrobic pyuria. The latter title is most widely used today. This disease has two outstanding features: No organisms can be isolated from the urinary sediment and the pyuria does not respond to the sulfonamides or the antibiotics.

ETIOLOGY

Five different possibilities have been presented by McGinn (*9*) as etiologic agents of this disease: (*a*) bacteria; (*b*) bacterial toxins; (*c*) *Treponema pallidum;* (*d*) fungi; and (*e*) viruses. McGinn and other observers believe that the etiological agent is probably an ultramicroscopic organism which falls into either the bacteria or the virus group. Although the bacterial theory receives the most recognition, such writers as Baines (*1*) and Moore (*10*) (*11*) favor an ultramicroscopic virus. Schauffhauser (*13*) has shown that the disease is easily transmitted from an affected human bladder to the kidney pelves of apparently normal dogs. Syphilis, toxins, and the fungi now receive little recognition as causes and are mentioned only for completeness.

SYMPTOMS

The outstanding symptoms are dysuria, diuria, nocturia, terminal pain, and terminal hematuria. Several or all of these symptoms may be present. In approximately 50 percent of the cases, nonspecific urethritis may usher in the syndrome. On physical examination suprapubic tenderness and urethral discharge may be the only positive findings. As the disease progresses, the symptoms increase in severity and opiates are often necessary to alleviate the pain over the bladder area. The patient presents no evidence of weight loss but usually has symptoms of a low-grade infection early in the disease. Later,

[1] U. S. Naval Hospital, St. Albans, Long Island, N. Y.

cachexia may occur as a result of the severe illness. A high fever is rarely found; usually it is relatively low grade. On occasion, a patient may have acute urinary retention because of the accumulation of enormous numbers of clots within the urinary bladder. Fortunately, such bladder hemorrhage is rare.

Laboratory examinations usually show a normal red blood cell count, unless extreme hematuria is present, a normal white blood cell count, and a normal differential count. The nonprotein nitrogen is within normal limits and the sedimentation rate is usually moderately elevated. Urinalysis usually shows from 1 to 4 plus albumin, negative sugar, 4 plus occult blood, and a sediment containing numerous leukocytes and red blood cells. Bacteria cannot be demonstrated in the urinary sediment. Cultures for *Mycobacterium tuberculosis* and other bacteria are negative, and the injection of guinea pigs with the urinary sediment fails to indicate any evidence of tuberculosis.

On cystoscopic examination, the bladder is extremely contracted, diffusely injected, and the mucosa covered, almost in its entirety, by scattered collections of purulent material. The ureteral orifices are visualized with difficulty and show distortion because of the edema and contraction of the bladder. A retrograde pyelogram usually shows minimal pyelectasis and ureterectasis. As the disease progresses, however, considerable dilatation of the ureters and kidney pelves occurs due to lower urinary tract obstruction. Solomon (*14*) states that all these changes in the upper urinary tract are reversible if proper treatment with arsenical preparations is undertaken early in the disease. In our case, no pathologic changes in the upper urinary tract were ever noted.

At present there is some controversy concerning recurrence of the disease. Moore (*10*) (*11*), in his observations, has found recurrences almost nonexistent although Landes (*7*) found them to be prevalent. Our patient showed no evidence of recurrence.

Several authors (Landes (*7*), Baines (*1*), and others) believe that Reiter's syndrome (which consists of nonspecific urethritis, conjunctivitis, and arthritis) and amicrobic pyuria are different manifestations of the same disease. They base their theory on the fact that patients who have what appears to be amicrobic pyuria, on occasions, show in rapid succession, the conjunctivitis and arthritis described by Reiter in his original paper. Therefore, they believe that both diseases are one and the same, appearing either in the mucous membranes of the eyes, the urethra, or joints.

TREATMENT AND THERAPEUTIC TEST

Neosalvarsan was formerly used in the treatment of this disease, but, because of the toxicity of this arsenical, the trend in this country

during the past decade has been to Mapharsen as the arsenical of choice.

A therapeutic test has been described by McGinn (*9*) and Landes (*7*), which they believe is extremely reliable in amicrobic pyuria. One injection of Mapharsen (usually 0.02 gm.) or some other arsenical is given; if the patient responds symptomatically within a few days, it is reasonable to suppose that the disease is amicrobic pyuria. Antibiotics as a rule have failed to bring any relief. In the United States, at present, Mapharsen is most widely used, while in the British Isles, Novarsenobenzol is the drug of choice, the dosage recommended being 0.3 gm. every 5 to 7 days for 4 doses. The dosage recommended for Mapharsen by the authors is an initial dose of 0.02 gm. and then 0.04 gm. 3 times a week for 9 doses. A symptomatic improvement follows this dosage in true amicrobic pyuria.

Reports of the use of streptomycin in the treatment of this disease are few, but Hamm (*5*) has reported some symptomatic relief with the drug.

CASE REPORT

An 18-year-old white man was admitted to the Naval Hospital, St. Albans on 3 November 1948, with the chief complaint of pain on urination. Present illness began 2 weeks before admission at which time (2 weeks after intercourse without prophylaxis) he noted severe burning at the end of urination and a moderate purulent discharge. One and a half weeks before admission, the pain at the end of urination had become very severe, dysuria was noted, and the discharge had become serous and "pinkish" in color. Symptoms were minimal at first, but as time progressed, the burning became more severe. He received treatment; at that time, urine examination showed a 1 to 2 plus albumin, 10 to 20 white blood cells per high power field, and an occasional red blood cell. It was then considered that he had acute cystitis (nonvenereal) and he was given sulfadiazine 1 gm. every 4 hours for 1 week. He was relatively asymptomatic for 2 to 3 days, when dysuria and diuria recurred and he was hospitalized.

Past and family histories and a review of systems were entirely within normal limits.

Physical examination revealed normal blood pressure, pulse, and respiration. The patient appeared depressed and chronically ill. Two large solitary lymph nodes were palpable in the left groin. The heart and lungs were within normal limits. A serous urethral discharge was present which showed no evidence of *Neisseria gonorrhoeae* on gram stain. Hemoglobin, 13 gm.; differential count normal; red and white blood cell count normal. Urinalysis showed acid urine, specific gravity, 1.024, 4 plus occult blood, 4 plus albumin, and numerous red blood cells and leukocytes. The three-glass test showed shreds and a cloudiness in all three specimens. On rectal examination the prostate was firm, nontender, and nonnodular. The prostatic secretions were within normal limits on repeated examinations. The Kahn test was reported as negative; the nonprotein nitrogen was 30.5 mg. per 100 cc. blood.

On 5 November 1948, a McCarthy panendoscope was passed without difficulty; the urethra showed no abnormality. The instrument was removed, and a No. 24 Brown-Buerger cystoscope was passed without difficulty. The urinary

bladder showed a distinct inflammatory reaction; the capacity was reduced to 50 cc. The left ureteral orifice could not be visualized because of severe inflammatory reaction on that side; the right orifice was catheterized after repeated attempts. Retrograde pyelogram showed no evidence of gross pathologic changes on the right side; an intravenous pyelogram on the same day revealed good function bilaterally with normal pelves, calyces, and ureters.

The diagnosis was changed on 6 November to acute nonvenereal cystitis.

Penicillin and sulfadiazine were administered immediately after cultures were taken. All cultures showed "no growth" after 48 hours; the cultures for tuberculosis showed "no growth" after several weeks. Penicillin, 100,000 units every 6 hours, and sulfadiazine, 1 gm. every 4 hours was administered. No alleviation of symptoms was obtained and the sulfadiazine was discontinued after 5 days and the penicillin after 6 days. Subsequently retreatment with penicillin and sulfadiazine gave no relief. Daily urinalyses showed no change in the pyuria and hematuria.

A cystogram on 15 November showed an irregular, contracted bladder with a capacity of 75 cc. and a small left bladder diverticulum measuring 4×3 cm. Bilateral retrograde pyelograms made on this date showed no gross pathologic change. Three days following this examination, the patient had a severe conjunctivitis which was considered to be "catarrhal conjunctivitis." Cultures of the eyes were negative for bacteria. The condition responded to symptomatic therapy and after 7 days no further evidence of conjunctivitis was found.

Repeated cultures of the urine for bacteria and acid-fast bacilli were negative. Symptomatic treatment was continued but the patient showed no improvement.

Cystoscopic examination on 9 December showed cystitis was still present and the bladder capacity was still only 100 cc. One week later, bladder capacity was found to be only 75 cc. although a few weeks later it was 125 cc. No residual urine was found at any examination.

On 4 February, because of the very slow symptomatic improvement Mapharsen was given. An initial dose of 0.02 gm. was given followed by nine triweekly doses of 0.04 gm.; a total of 0.38 gm. was given. During the period of treatment, the bladder capacity increased greatly and symptomatic relief was appreciable. At the end of treatment, the bladder capacity was 375 cc., urinalyses were entirely within normal limits, and the patient was completely asymptomatic.

Cystoscopic examinations on 4 March 1949, revealed a normal-appearing bladder with the exception of the left-sided diverticulum. The latter showed no evidence of increase in size. The bladder capacity at this time was 425 cc. There has been no evidence of recurrence to 15 December 1949.

REFERENCES

1. BAINES, G. H.: Relation of abacterial pyuria to Reiter's syndrome. Brit. M. J. 605–608, Oct. 18, 1947.
2. BRIGGS, W. T.: Etiology and therapy of non-tuberculous kidney infections. Urol. & Cutan. Rev. **39**: 149–155, March 1935.
3. COOK, E. N.: Infections of urinary tract without demonstrable organisms. Proc. Staff. Meet., Mayo Clin. **19**: 377–380, July 26, 1944.
4. COOK, E. N.: Infections of urinary tract of obscure etiology. J. Urol. **36**: 460–465, Oct. 1936.
5. HAMM, F. C.: Amicrobic pyuria. J. Urol. **57**: 226–232, Feb. 1947.
6. HOUTAPPEL, H. C. E.: Sterile pyuria. Urol. & Cutan. Rev. **43**: 477–479, 1939.
7. LANDES, R. R. and RANSOM, C. L.: Abacterial pyuria; possible relationship to Reiter's syndrome. J Urol. **60**: 666–677, Oct. 1948.

8. LYDON, F. L.: Reiter's syndrome and abacterial pyuria. Brit. M. J. 974–975, Dec. 13, 1947.
9. MCGINN, E. J.: True infective abacetrial pyuria. Wisconsin M. J. 45: 845–847, Sept. 1946.
10. MOORE, T. (Manchester): True infective abacterial pyuria. Brit. J. Urol. 17: 131–135, Dec. 1945.
11. MOORE, T.: Abacterial pyuria. Brit. M. J. 1: 170–176, 1940.
12. PETERS, W.: Aseptic renal pyuria. Abstr. J. A. M. A. 24: 1750–1752, 1920.
13. SCHAUFFHAUSER, F.: Die Sogennanten abaklenellen renalen pyurien. Ztschr. f. Urol. Chir. u. Gynak. 43: 83–140, 1937.
14. SOLOMON, A. A.: Effect of arsenotherapy on upper urinary tract changes in infectious abacterial pyuria. J. Urol. 59: 252–257, Feb. 1948.
15. WALLMAN, D. R.: Report of case of true infective abacterial pyuria. M. J. Australia 2: 93–94, July 20, 1946.

866046°—50——6

Exfoliative Dermatitis Following the Administration of Penicillin

EDWARD LANGDON, *Captain, MC, A. U. S.*[1]

THE increasing use of penicillin in the past 5 years has been accompanied by numerous reports of adverse reactions. One of the less frequently described types of reaction has been generalized exfoliative dermatitis.

Nolan and Pedigo (*1*) found no report in the literature prior to April 1946, of exfoliative dermatitis following the administration of penicillin, and reported the case of a 60-year-old man who developed urticaria and generalized erythema after 75,000 units of his first course of penicillin. Two weeks later penicillin therapy was reinstituted. After 80,000 units the erythema recurred and progressed to a generalized exfoliative dermatitis. After recovery a patch test was positive for the brand of penicillin used, but was negative with another brand. Goldman et al. (*2*), while reporting various dermatoses from penicillin, mentioned the exfoliative type in a 50-year-old man. The dermatitis appeared after 1.9 units of penicillin had been given intramuscularly and 80,000 units had been given intrathecally. A positive skin reaction to penicillin was demonstrated after recovery. Eilert (*3*) reported a case of exfoliative dermatitis with spontaneous recovery and a positive intradermal test in a 56-year-old man receiving an aqueous solution of penicillin intramuscularly.

Farrington and Tamura (*4*) reported the case of a 78-year-old man who received an aqueous solution of penicillin intramuscularly. On the fourth day of treatment he developed generalized erythematous maculopapular eruption. After three additional days of penicillin therapy vesicular and bullous lesions appeared over the thighs, buttocks, and abdomen, and the erythema became confluent. Therapy was discontinued, but exfoliation of the skin persisted with loss of the nails and slight alopecia. Positive skin reactions to penicillin were observed after recovery. Shaffer (*5*) reported a 27-year-old man who developed exfoliative dermatitis after having received peni-

[1] Fitzsimons General Hospital, Denver, Colo.

cillin locally and intramuscularly. No skin tests were made. Barksdale (*6*) reported the case of a 52-year-old man with stasis dermatitis of the legs and fungus infection of the feet complicated by cellulitis; this patient developed generalized vesicular dermatitis while receiving penicillin locally and intramuscularly. One month later a penicillin patch test was negative. Nine days of treatment with penicillin locally produced no adverse effects, and aqueous penicillin was given intramuscularly at the rate of 1,000 units every 6 hours. Seventeen hours later the patient experienced severe generalized itching. Penicillin was discontinued after the fifth dose. After 5 days of intramuscular injections of penicillin the patient developed a generalized exfoliative dermatitis. His condition was complicated by a staphylococcal septicemia. Death occurred 20 days after penicillin had been discontinued. Autopsy revealed interstitial hepatitis, interstitial nephritis, and exfoliative dermatitis. Staphylococcal septicemia was considered to be the cause of death.

Two cases of exfoliative dermatitis following the administration of penicillin are reported.

CASE REPORTS

Case 1.—A 23-year-old man was admitted on 18 October 1948. He had received daily intramuscular injections of penicillin in oil and beeswax and aqueous penicillin locally for trench mouth from 24 to 28 September. One week after therapy had been discontinued he noticed a pale erythematous pruritic eruption in the intercrural folds. For the following week he was treated with Benadryl locally without relief. The erythematous eruption became generalized and was followed by exfoliation. The process involved the scalp and face, but not the palms and soles. His only complaints were pruritis and sensations of chilliness. Physical examination revealed generalized exfoliation of fine white scales from a slightly erythematous base and a mild nontender generalized lymphadenopathy. The leukocyte count was 7,000 with 55 percent neutrophils, 40 percent lymphocytes, 3 percent monocytes, 1 percent eosinophils, and 1 percent basophils. The patient remained in the hospital for 1 week. Therapy consisted of constant dusting with purified talc. At the time of discharge he had a slight residual exfoliation but was asymptomatic.

Case 2.—A 53-year-old man was admitted on 23 December 1948. He gave a history of having received daily intramuscular injections of penicillin in an absorption-delaying vehicle on 24 and 25 November for a furuncle on the neck. Four days later he noticed an itching erythematous rash in the groin and urticaria over the abdomen. In the following week the erythema became generalized and the epidermis began to exfoliate in large flakes. This process continued for 2 weeks, during which time his only symptom was severe pruritus. Three days before admission he became anuric, slightly dyspneic, nauseated, and vomited several times daily. Past history revealed that albuminuria had been noted in 1944, and that he had been retired from the Armed Forces in 1946 with a diagnosis of nephritis. His complaints prior to those of the present illness were minimal ankle edema after prolonged standing and nocturia one or two times nightly.

Physical examination revealed a severe generalized exfoliative dermatitis involving the scalp, face, palms, and soles. The skin was deeply erythematous and slightly edematous. There were multiple small weeping fissures about the forearms. The furuncle of the neck had healed. The conjunctival sacs contained small amounts of yellow exudate. The retinal arterioles were moderately narrowed with slight arteriovenous nicking. There was a large patch of exudate lateral to the right optic disk. The blood pressure was 180/85; nonprotein nitrogen, 320 mg. per 100 cc. blood; creatinine, 11.5 mg. per 100 cc. blood; and CO_2 combining power, 29 volumes per 100 cc. blood. The hemoglobin was 11.4 gm.; erythrocyte count, 3,920,000; and leukocyte count, 19,000 with 72 percent neutrophils, 15 percent lymphocytes, 8 percent monocytes, and 5 percent eosinophils. The specific gravity of the urine was 1.008. The urinary albumin was 168 mg. per 100 cc.

Local therapy with powdered corn starch was begun, but was later changed to olive oil application when fissuring became more extensive. He was given a 2,000 calorie diet containing 40 gm. of protein. The fluid intake was maintained at about 3 liters daily. On the second hospital day he began to excrete urine, but the volume remained small. His general condition remained stable until the tenth hospital day when there was a recurrence of nausea and vomiting. He became drowsy. Additional therapy included intravenous infusions of M/6 sodium lactate, a blood transfusion, calcium gluconate, and crude liver extract intramuscularly. He was slightly improved on 6 January 1949, but on the following morning, while being helped out of bed, he became unconscious and cyanotic and died 20 minutes later. During the hospital course the nonprotein nitrogen decreased irregularly to 186 mg. per 100 cc. blood on the day preceding death. The creatinine varied from 11.2 to 14.8 mg. per 100 cc. blood. The CO_2 combining power varied between 14 and 33 volumes per 100 cc. blood. On 4 January the calcium was 6.7 mg. per 100 cc. blood. The specific gravity of the urine never exceeded 1.009. The urinary albumin varied from 0 to 168 mg. per 100 cc. The hemoglobin and erythrocyte counts gradually declined to 9.4 gm. per 100 cc. and 3,050,000, respectively. The electrocardiogram showed sinus tachycardia, left axis deviation, and a prolonged Q–T interval. Retrograde pyelography revealed small contracted kidneys bilaterally.

In addition to severe exfoliative dermatitis, post-mortem examination revealed the changes of chronic glomerulonephritis with kidneys weighing 65 and 74 gm. The cortices were 0.3 cm. in diameter and the boundaries between cortex and medulla were difficult to distinguish. The glomeruli presented various stages of degeneration from increased cellularity and decreased vascularity to replacement by fibrous tissue and hyalinization. The process in some of the glomeruli was thought to be of recent origin. The liver weighed 2,140 gm. and showed destruction of the normal architecture with central fatty change. The heart weighed 400 gm. The ventricular walls were not obviously hypertrophied, but there was some enlargement of the papillary muscles of the left ventricle and thickening of the chordae tendinae of the mitral valve. There was moderate chronic passive congestion of the lungs, liver, spleen, and kidneys. The mucous membrane of the bladder was denuded. Death was caused by chronic glomerulonephritis and exfoliative dermatitis.

DISCUSSION

It is surprising that so few cases of exfoliative dermatitis following the administration of penicillin have been reported since many more must have occurred. Apparently, there are several factors that, alone

or in combination, predispose to dermatitis; these are penicillin, its impurities, the vehicle, and extrinsic sources of fungi. Sensitization may follow contact, ingestion, inhalation, or injection of these agents. All of the cases reported have been in men; their ages ranging from 23 to 78 years. In both of the patients who died, renal and hepatic damage was found at autopsy. In one there was a history and evidence of preexisting chronic glomerulonephritis, but in addition, there were acute lesions in some of the glomeruli. Because of the possibility of severe exfoliative dermatitis, penicillin should be used only in the presence of definite indications.

References

1. Nolan, D. E., and Pedigo, G. W., Jr.: Exfoliative dermatitis following penicillin therapy. Ann. Int. Med. **25**: 725–728, Oct. 1946.
2. Goldman, L.; Friend, F.; and Mason, L. M.: Dermatitis from penicillin. J. A. M. A. **131**: 883–890, July 13, 1946.
3. Eilert, M. L.: Symposium on advances in clinical medicine; adverse reactions to sulfonamides and antibiotics. M. Clin. North America **31**: 31–42, Jan. 1947.
4. Farrington, J., and Tamura, J.: Cutaneous testing in case of exfoliative dermatitis caused by penicillin. Arch. Dermat. & Syph. **56**: 807–811, Dec. 1947.
5. Shaffer, J. O.: Generalized exfoliative dermatitis due to penicillin. New England J. Med. **238**: 660–661, May 6, 1948.
6. Barksdale E. E.; Frost, D. M.; and Nolan, J. J.: Reactions from penicillin. U. S. Nav. M. Bull. **48**: 883–886, Nov.–Dec. 1948.

Acute Porphyria

Report of a Case With Post-mortem Findings

ROGER H. FULLER, *Lieutenant Commander (MC)* U. S. N.[1]

ASIDE from the identification of the abnormal porphyrin compounds in the tissues or body fluids by chemical or physical means, there are no pathologic criteria by which a post-mortem diagnosis of porphyria may be made with certainty. However, the metabolic abnormality is, if long enough continued, accompanied by morbid anatomic changes which are detectable by routine pathologic methods. Although the findings are not specific, they may confirm the clinical or chemical diagnosis or may suggest the proper direction for investigation if a diagnosis has not been previously made. Furthermore, these anatomic expressions of disease shed some light on the varied symptoms.

Clinical diagnoses of porphyria are made much more frequently now than heretofore, and the disease is no longer considered a rarity. However, the descriptions of the post-mortem findings are few. For this reason, and because it shows a combination of congenital metabolic and congenital anatomic defects not previously noted, this case is reported.

CASE REPORT

Clinical history.—A 26-year-old white woman was well until July 1947 when there was an acute onset of abdominal colicky pain with nausea and vomiting. A diagnosis of intestinal obstruction was made, but was not found at operation. Following the operation she had severe weakness of the extremities; her appetite was poor; and during the succeeding 4 months her weight decreased from 112 to 95 pounds. In November 1947 she experienced a second episode of acute abdominal pain and was hospitalized for 4 days without a diagnosis or specific treatment. Following discharge there was a gradual onset of muscular paralysis beginning in the legs and ascending to involve the trunk and arms. There was also bladder and rectal incontinence. However, after about 6 weeks, function gradually returned and by June 1948 she was able to do her housework.

On 8 August 1948 she experienced her third attack of cramping abdominal pain with vomiting. On 12 August she began to have pain in the back of her legs and numbness in her arms. She was admitted to the Henry Ford Hospital.

[1] Department of Pathology, Henry Ford Hospital, Detroit, Mich.

Physical examination revealed an emaciated young woman complaining of considerable abdominal pain. The abdomen was not tender or rigid; no masses or solid organs were palpable. Reflexes were hypoactive throughout. There was wasting of the musculature, most noticeable over the thighs and in the intrinsic muscles of the hands. The patient could walk, but staggered. The admission diagnosis was hysteria.

The pain left the legs quite suddenly, but was intensified in the abdomen. Sodium amytal narcosis was instituted on 14 August and maintained through 17 August, when it was noted that the amytal effect was excessively prolonged, and a damaged liver was suspected. On 18 August cephalin cholesterol was 3 plus; thymol turbidity, 3 units; and thymol flocculation, 4 plus.

While the patient was narcotized, it was learned from the family that the patient's brother had died in 1946 at the age of 26 of an undiagnosed disease causing paralysis of his upper arms, generalized weakness, and abdominal pain. An autopsy was performed and death was attributed to pneumonia. Physicians told the mother that a conclusive diagnosis was never made.

On 18 August the patient, free of narcosis, began to have both visual and auditory hallucinations. She was seen by the neurologist on 21 August, and a diagnosis of acute porphyria was suggested. Watson's test for porphobilinogen was positive. Uroporphyrin was identified in the urine by spectrophotometric methods. The urine was clear and amber when voided, but became a port wine color after standing 24 hours.

The weakness and flaccid paralysis increased, and signs of bulbar paralysis became evident. Blood pressure which had been about 158/110 fell to 118/68; she showed evidence of consolidation of the lower lobe of the right lung and death followed.

Post-mortem Findings

The gross post-mortem findings of note consisted of a severe degree of emaciation with muscle atrophy in the lower extremities. There was a confluent bronchopneumonia in the right lung and early bronchopneumonia in the dependent portions of the left lung. The liver weighed 1,280 grams and appeared normal. The kidneys contained many cysts up to 4 cm. in diameter, were somewhat enlarged, and appeared fairly typical of polycystic disease with large areas of functioning kidney tissue between the cysts. The gross appearance of the brain and spinal cord was normal except that the gray matter of the lumbosacral cord appeared hyperemic. Sections of spinal cord and brain exposed to sunlight did not develop any unusual pigment. The peripheral nerves were grossly normal.

Microscopic examination.—The sections of the liver showed some increase in nuclei in the periportal areas with enlargement of these areas and some slight collagen deposition. The hepatic cells presented no abnormalities. No effort was made to demonstrate fluorescent material in the cells. The lungs showed confluent bronchopneumonia in the right and early bronchopneumonia in the left. In the kidneys there were many thin-walled cysts of various sizes scattered through the cortex and the zone between cortex and medulla. The intervening kidney parenchyma showed no change except those due to pressure. Adrenals showed no significant change. Sections of other thoracic, abdominal, and pelvic organs showed nothing unusual in the routine hematoxylin and eosin stain.

Nervous system.—Sections of brain, spinal cord, peripheral nerve, and celiac ganglion were stained with hematoxylin and eosin, Weigert's method for myelin sheaths, and Bodian's stain for axis cylinders. Fixation of the spinal cord was especially good, and it was in the lumbar and sacral areas of the spinal cord

that the most distinct changes were found (fig. 1). These consisted of alterations in the anterior horn cells similar to those reported by others. There was perinuclear chromatolysis spreading to involve all the Nissl substance in the cell, swelling of the cell body, lysis of nucleus, eccentric position of the nucleus, vacuolization of the cytoplasm, and shrinkage and increased affinity of the cell for basic stain. In the lower spinal cord almost all the anterior horn neurones were affected to some degree, although a few appeared perfectly normal and served as a control in assessing post-mortem changes. Changes in the brain stem and cortex were difficult to evaluate because of the fact that fixation was not as rapid as in the case of the spinal cord. No demyelinization was demonstrated in the brain or spinal cord. There was no glial reaction noted, nor were there exudative phenomena.

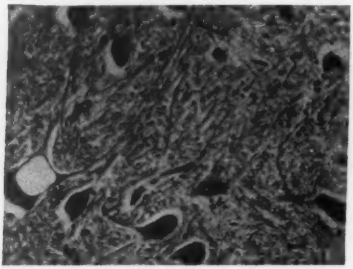


Figure 1.—Anterior horn of lumbar cord showing degenerative changes in the neurones. Note chromatolysis, lysis of nuclei, and the huge vacuole in the cytoplasm of one cell. Hematoxylin and eosin stain.

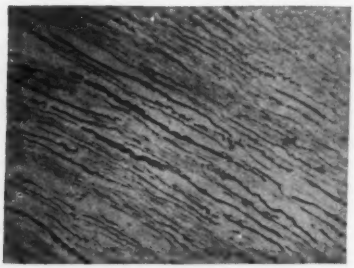


Figure 2.—Peripheral nerve of lumbar plexus showing swelling, tortuosity, and fragmentation of the axones. Bodian stain for axis cylinders.

In the peripheral nerves, alteration in the axis cylinders and myelin sheaths was found in varying degrees of severity and patchy distribution (fig. 2). Vacuolization and breaking up of myelin into large globules; swelling, tortuosity, and fragmentation of axis cylinders could be demonstrated. In addition, there was edema fluid beneath the perineurium. A scanty infiltration of nerves by vacuolated wandering cells was occasionally seen. Sections of the celiac ganglion, when compared with sections of normal ganglion from patients of the same age, showed an increased amount of pigment in the cytoplasm of the neurones.

Comparison of the neuronal changes in the spinal cord with those in a spinal cord from a case of amyotrophic lateral sclerosis revealed many similarities. These neuronal changes were also similar to those produced in experimental anoxia by Hartman (*1*), Denny-Brown (*2*), and others.

The pathologic diagnoses were peripheral neuropathy consistent with acute porphyria, bronchopneumonia, and congenital polycystic kidneys.

COMMENT

The pathologic changes in the nervous system in this case are essentially the same as those found by Denny-Brown and Sciarra (*3*), Courville and Mason (*4*), (*5*), and others (*6*). Some workers have

described focal areas of necrosis in the liver and kidneys in fatal cases of acute porphyria; others have demonstrated by means of ultraviolet microscopy fluorescent compounds in the hepatic, renal, and nerve cells. Berg (*7*) in his study of a patient during an attack of acute porphyria noted spasm of the fundal arteries, capillary spasm, and aneurysmal dilatation in the skin and nail beds. A transient vascular hypertension is usually present during acute attacks, suggesting an element of vasospasm. It has been suggested that vasospasm, caused by the abnormal porphyrin compound or its precursor, causes tissue anoxia and is responsible for the patchy lesions found.

References

1. Hartman, F.: Some etiological factors and lesions in cerebral anoxia. Am. J. Clin. Path. **8**: 629, 1938.
2. Denny-Brown, D., and Brenner, C.: Paralysis of nerve induced by direct pressure and by tourniquet. Arch. Neurol. & Psychiat. **51**: 1, 1944.
3. Denny-Brown, D., and Sciarra, D.: Changes in central nervous system in acute porphyria. Brain **68**: 1, 1945.
4. Courville, C., and Mason, V. R.: Acute ascending paralysis (Landry's Paralysis) with acute idiopathic hematoporphyrinuria. Arch. Neurol. & Psychiat. **25**: 848, 1931.
5. Mason, V. R.; Courville, C.; and Ziskind, E.: Porphyrins in human disease. Medicine **12**: 355, 1933.
6. Baker, A. B., and Watson, C. J.: Central nervous system in porphyria. J. Neuropath. & Exper. Neurol. **4**: 68, 1945.
7. Berg, M.: Acute porphyria; clinical and pathologic observations. Arch. Int. Med. **76**: 335, 1945.
8. Nesbitt, S., and Watkins, C.: Acute porphyria. Am. J. M. Sc. **203**: 74, 1942.

Epidural Abscess Secondary to Brucellosis ("Brucella Suis")

JOHN W. SUMNER, JR., *Major, MC, U. S. A.*[1]

TO DETERMINE the etiologic factors in low back pain or headache often tests the ingenuity of the physician; in many patients no pathologic changes can be found. The patient suffering from undiagnosed brucellosis is often regarded as hypochondriacal, so protean are its manifestations. Arthralgia involving any or all joints is a common symptom of brucellosis but abnormal roentgenographic findings are usually absent except in instances of spondylitis and sacroiliac arthritis. Phalen et al. (*1*) emphasize that lumbar spondylitis is a common manifestation of brucellosis and report three patients in two of whom destructive lesions in the lumbar vertebrae were revealed by roentgenogram. Steinberg (*2*) reported a case of left sacroiliac arthritis secondary to brucellosis in which roentgenograms showed destructive bone changes in the left sacroiliac joint. In 1932 Kulowski and Vinke (*3*) cultured *Brucella* organisms from a large abscess in the region of the right transverse processes of the lower three lumbar vertebrae. This was the first proved instance of a suppurative lesion in the spinal column secondary to brucellosis.

CASE REPORT

A 29-year-old Negro was admitted to this hospital on 22 April 1948 complaining of recurrent low back pain, fever, and night sweats. He had been discharged from the Army in April 1946. He denied ever working with cattle or swine before 1946. In November 1946 he got a job cleaning hogs' intestines for 2 months; during this period he occasionally cut his fingers but had no evidence of local infection; in January 1947 he was handling hogs' livers. He wore no gloves while at either of these jobs. In March 1947 he was removing and cleaning cows' rumens. He occasionally cut his hands while at this work. In the latter part of December, while working, a bone spicule penetrated his right forearm to a depth of about 1 inch; the injury healed uneventfully. In January 1948 he sustained a small laceration of the skin of his left upper arm when he ran into a door. He applied a disinfectant to the wound, but did not bandage it, and continued his work. In the third week in March he suddenly began

[1] Fitzsimons General Hospital, Denver, Colo.

vomiting and in the next week he had mild low back pain; a few days later he had fever and profuse sweating. A physician gave him sulfonamide tablets for 1 week and then recommended hospitalization. The admission diagnosis was back strain.

On admission his temperature was 101.6° F., pulse, 104, and respiration, 22. There was spasm of the right sacrospinalis muscle and moderate tenderness on palpation in this region. He was treated by rest on a hard bed, massage, radiant heat, and passive exercises of the back muscles. Improvement was noted and by 26 April he was completely asymptomatic and afebrile. He was discharged with a diagnosis of cured acute moderate myositis of the right sacrospinalis muscle, cause undetermined.

He was readmitted on 17 May complaining of recurrence of low back pain, profuse sweating, fever, and loss of weight. The low back pain was dull and constant, but occasionally became sharp and radiated laterally to the right iliac crest, then antero-medially into the inguinal region. There was no history of his ever having drunk raw milk.

On admission his temperature was 102.2° F., pulse, 94, and respiration, 18. He had lost 20 pounds in weight. Several shotlike lymph nodes were palpable in the right inguinal region. Examination of the back revealed tenderness and moderate spasm of the left paravertebral muscles in the lumbar area. Straight-leg raising on the left was limited by pain in the left lumbar area. Pain on percussion was present in the costovertebral angle on both sides. Tentative diagnoses of tuberculous spondylitis and pyelonephritis were made. Initial laboratory data revealed hemoglobin, 13.2 gm.; a leukocyte count of 4,200 with 74 percent neutrophils, 23 percent lymphocytes, 1 percent monocytes, and 2 percent eosinophils. The sedimentation rate was 22 mm. (Wintrobe). A roentgenogram of the chest showed a moderate increase in the lung markings in the first and second anterior interspaces bilaterally, but no infiltration was demonstrated and a roentgenogram of the lumbar spine showed slight scoliosis to the right.

On 19 May treatment with sulfadiazine, 1 gm. every 4 hours, and penicillin, 100,000 units every 3 hours, was started. The patient's fever continued for the next 6 days during which time there was no leukocytosis, but the sedimentation rate remained elevated. On 27 May chemotherapy was discontinued because the patient had been afebrile for 4 days and was symptomatically improved except for mild lethargy. On 2 June blood agglutinations for typhoid and paratyphoid were negative, but the blood agglutination for brucellosis was positive in a titer of 1:320. On 9 June, because of stiffness in the lumbar area, radiant heat to the lumbar area followed by back-strengthening exercises were started. On 24 June the patient became febrile again and complained bitterly of low back pain. Physical therapy was discontinued and chemotherapy was resumed. The patient became afebrile early in July, but the low back pain continued and weakness of the dorsiflexor muscles of both feet was noted. Chemotherapy was discontinued and he was placed in flexion traction for 1 week without relief. On 16 July a lumbosacral manipulation under pentothal sodium was performed. This gave almost complete relief of the low back pain. A lumbar puncture revealed 10 white blood cells, chlorides, 680 mg. per 100 cc., positive globulin, total protein, 190 mg. per 100 cc., and a colloidal gold curve of 5555555433. Spinal fluid examination on 28 July revealed 129 white blood cells, positive globulin, total protein, 334 mg. per 100 cc., gold curve, 5555543210, and a negative serologic test for syphilis. Because of progressive neurologic signs in the lower extremities and the abnormal spinal fluid findings, the patient was transferred to the neurologic section on 31 July with a tentative diagnosis of infectious polyneuritis or epidural abscess in the lumbar region.

Examination after transfer showed that the patient was only able to stand long enough to get from his bed to a wheel chair. There was atrophy of the thigh and calf muscles, more noticeable on the left, with bilateral weakness of the glutaei, gastrocnemii, extensor digitorum longi, and the peronaei muscles. The right tibialis anterior muscle was fairly strong but the left was weak. The external hamstring reflexes were sluggish, and the internal hamstring reflexes and ankle jerks were absent. There were no sensory changes except a diminution of vibratory sense in the left big toe.

The patient continued to complain of low back pain and night sweats and his fever fluctuated between 99° and 101° F. On 6 August his temperature went up to 102.8° F. Chemotherapy was again administered. Blood culture was negative, but the brucellosis agglutination was positive in a titer of 1:2560. The leukocyte count was 11,800 with 71 percent neutrophils, 28 percent lymphocytes, and 1 percent monocytes. The sedimentation rate was 49. Reexamination of the patient revealed a moderately enlarged spleen. On 12 August a lumbar puncture was attempted between the third and fourth lumbar vertebrae, but no fluid was obtained even after aspiration. Another needle was then inserted between the second and third lumbar vertebrae with the return of clear spinal fluid under a pressure of 160 mm. water. A third needle was inserted between the fifth lumbar vertebra and the sacrum. The dural sac was not entered in this region, for prior to reaching the depth of the dural sac a creamy exudate was recovered through the needle. On 13 August penicillin was discontinued and streptomycin, 0.5 gm. every 4 hours for 10 days together with sulfadiazine, 1 gm. every 4 hours, was given. Examination of the spinal fluid obtained from between the second and third lumbar vertebrae on 12 August revealed 37 white blood cells, one of which was a neutrophil, positive globulin, total protein, 210 mg. per 100 cc., and a gold curve of 5555543211. Roentgenograms of the lumbar spine on 14 August were negative for evidence of bone disease.

On 16 August a laminectomy was performed. The spinous process of the fifth lumbar vertebra was removed and the dura mater exposed, but no frank pus was encountered. The spinous process of the fourth lumbar vertebra was then removed and a small amount of necrotic material was found in the area of the fifth lumbar vertebra posteriorly between the neural arch and the dura. Further exploration revealed no additional exudate. At the operative site there was some bulging of the dura but it was not opened. The cauda equina, seen through the dura, appeared normal. The patient made a good recovery.

On 2 September the laboratory reported that cultures of the blood taken on 6 August and the creamy exudate discovered between the fifth lumbar vertebra and the sacrum on 12 August both revealed *Brucella suis*, confirmed serologically. Culture of the exudate taken at the time of the laminectomy after beginning treatment with streptomycin and sulfadiazine was negative. At discharge on 24 November the patient's only complaint was a slight weakness in dorsiflexion of the left foot: he was afebrile and the spleen could not be palpated. On neurologic examination, 50 to 80 percent of normal strength was noted in the muscles supplied by the left common peroneal nerve.

SUMMARY

An epidural abscess in the region of the fifth lumbar vertebra occurred in a patient who worked in a meat-packing plant. The etiologic factor did not become evident until after he showed abnormal neurologic signs in the lower extremities, abnormal spinal fluid findings, an enlarged spleen, and a brucellosis agglutination titer that rose

from 1:320 to 1:2560. Roentgenograms of the lumbar vertebrae were negative. Blood culture and culture of the epidural abscess were positive for *Brucella suis*. Drainage of the epidural abscess by laminectomy and treatment with streptomycin and sulfadiazine resulted in a cure.

References

1. Phalen, G. S.; Prickman, L. E.; and Krusen, F. H.: Brucellosis spondylitis; treatment by physically induced hyperpyrexia. J. A. M. A. 118: 859, March 14, 1942.
2. Steinberg, C. L.: Brucellosis as cause of sacroiliac arthritis. J. A. M. A. 138: 1519, Sept. 4, 1948.
3. Kulowski, J., and Vinke, T. A.: Undulant (Malta) fever spondylitis; report of case due to *Brucella melitensis*, bovine variety, surgically treated. J. A. M. A. 99: 1656–1659, Nov. 12, 1932.

Herpes Zoster With Concurrent Encephalitis

Report of a Case

ARVIN T. HENDERSON, *Lieutenant (MC) U. S. N.*
DONALD C. YOUNG, *Commander (MC) U. S. N. R.*
GEORGE B. KEMPTON, *M. D.*

THE skin lesions of herpes zoster are accompanied by inflammatory changes in the dorsal root ganglion and there is frequently an associated pleocytosis of lymphocytes in the spinal fluid. Only rare cases of involvement of the higher nerve centers are recorded, although this is not uncommon in animals where it has been produced experimentally (*1*).

CASE REPORT

K. M., a 17-year-old white girl, was admitted to the Communicable Disease Service of Herman Kiefer Hospital on 11 December 1948 complaining of severe frontal headaches and lethargy.

She was well until 2 weeks prior to admission when she noticed a localized, painful rash over the right side of the chest. Nine days later a dull frontal headache associated with faintness and dizziness was noted. With bed rest these symptoms subsided only to return with greater intensity the next day, accompanied by intermittent dull abdominal pain. All symptoms persisted and 3 days prior to admission she became nauseated, vomiting 3 times. When the headache became more severe she consulted her family physician who admitted her to a general hospital. The following morning a spinal puncture was performed which showed a pleocytosis with increased pressure and increased protein. She was given 300,000 units of penicillin intramuscularly and 100,000 units intravenously and then sent to this hospital for further observation.

The family history and past history were noncontributory.

Physical examination revealed a well-developed, well-nourished white girl who appeared acutely ill. Temperature, 99.8° F. per rectum, pulse 60, and respirations 18. She was very lethargic, irritable, moderately disoriented, and obeyed commands slowly. There was a discrete maculopapular skin rash over the lower one-third of the right scapula and the right axilla and extending medially to the right breast. Surrounding each of these lesions was a small area of erythema. Considerable nuchal rigidity and bilateral hamstring spasm was present. No muscular weakness was found; the tendon and superficial reflexes were hyperactive and equal. An abortive clonus was present on the right. The funduscopic examination and the remainder of the physical examination were essentially negative.

Laboratory findings.—Spinal fluid was slightly hazy with 496 lymphocytes per cu. mm.; sugar, 48 mg.; and protein, 154.6 mg. per 100 cc. There was no growth of organisms on culture. Red and white blood cell count, normal; hemoglobin, 80 percent; differential count normal; and 3 percent eosinophils. Blood Kahn test was negative.

Hospital course.—The temperature remained normal throughout her hospital stay. The central nervous system symptoms decreased considerably following the spinal tap. Intravenous fluids were given the first day, but nourishment was taken by mouth thereafter. On the second hospital day, the patient was much improved, bright, and well-oriented. Her nuchal rigidity had diminished, and the ankle clonus had disappeared. On the third day, improvement continued, but lesions on the thoracic wall had increased in number and the erythema had become confluent. The lesions followed the distribution of the fourth and fifth intercostal nerves on the right side ending sharply at the midline. Vesiculation and crusting were present. A spinal tap revealed 489 lymphocytes per cu. mm.; sugar, 48 mg.; chlorides, 685 mg.; protein, 110 mg. per 100 cc. She continued to improve, and on the sixth hospital day all neurological signs and symptoms had disappeared and the skin lesions had improved. Spinal fluid at this time showed 180 lymphocytes per cu. mm.; sugar, 43 mg.; protein, 109 mg.; and chlorides, 707 mg. per 100 cc. She was discharged from the hospital. One and a half weeks after leaving the hospital the skin lesions had disappeared completely.

DISCUSSION

We believe this to be a case of herpes zoster with meningoencephalitis. There have been a few cases reported in the literature with similar clinical findings, and necropsies were performed upon some.

The typical case ((*2*) (*3*) (*4*) (*5*) (*6*) (*7*) (*8*) (*9*) (*10*)) is characterized by the appearance of lesions of herpes zoster followed in 1 to 4 weeks by the symptoms of encephalitis, prominent among which are headache, drowsiness, nausea, vomiting, disorientation, weakness, paralysis (peripheral and cranial), coma, and sensory disturbances. Although there are few reports of spinal fluid findings, there was characteristically a lymphocytosis, increased globulin, and a normal sugar content. The autopsy findings are those of herpes zoster involving the dorsal root or extra-medullary cranial nerve ganglia with extension into the spinal cord and brain stem; of perivascular small, round cell infiltration; hyperemia, hemorrhages, and neuroglial proliferation. Involvement of the cerebrum is minimal.

In this case, the herpes zoster preceded the onset of central nervous system symptoms (headache, vertigo, nausea, and vomiting) by 1 week. Neurological findings consisted of lethargy, irritability, nuchal rigidity, and hamstring spasm. The deep and superficial tendon reflexes were hyperactive, and there was abortive clonus on the right. The spinal fluid findings were compatible with encephalitis, as was the rapid recovery.

A diagnosis of herpes zoster concomitant with lymphocytic choriomeningitis cannot be ruled out, since neither pathological studies, facilities for isolation of the virus, nor serum antibody studies were

available in this case. The possibility of polioencephalitis should be noted; however, this case occurred in December, and the signs and symptoms were not indicative of this disease. Tuberculous meningitis was considered, but discounted after a negative tuberculin test, negative roentgenogram of the chest, spinal fluid findings which were not compatible with this type of meningitis, and recovery. Syphilitic meningitis was disproved by negative serologic studies. Netter (*11*) found that when herpes zoster and epidemic encephalitis occurred in the same patient, zoster followed encephalitis.

Krumholz et al. (*10*) interpreted the pathologic changes to suggest a toxic encephalitis due to an allergic response of the patient to the virus of herpes zoster. Our spinal fluid findings on three occasions of high cell count and high globulin suggest an extension of herpetic infection rather than a toxic or allergic manifestation.

SUMMARY

Herpes zoster was followed in 1 week by manifestations of encephalitis in a 17-year-old girl. It is suggested that the etiological agent was common to both.

REFERENCES

1. HASSIN, G. B.: Herpetic meningoencephalitis. J. Neuropath. & Exp. Neurol. **3**: 355–367, 1938.
2. THALHIMER, W.: Herpes zoster: central nervous lesions similar to those of epidemic encephalitis. Arch. Neurol. & Psychiat. **12**: 73, 1924.
3. BIGGART, J. H., and FISHER, J. A.: Meningoencephalitis complicating herpes zoster. Lancet **2**: 944, 1938.
4. ANDERSON, M. S., and WULFF, F.: Meningitis serosa und encephalitis bei zoster. Acta. psychiat. et neurol. **8**: 213, 1933.
5. BELLAVITIS, A.: Nevrassite da herpes-zoster. Contribute clinicoanatomico. Riv. neurol. **4**: 337, 1931.
6. ROGER, H.: POURSINES, Y.; and PAILLAS, J.: Orinine zosterienne probable d'une poliomesencephalo-myelite subsique. Rev. otoneuro-oftal. **16**: 176, 1938.
7. SCHIFF, C. I., and BRAIN, W. R.: Acute meningoencephalitis associated with herpes zoster; a fatal case. Lancet **2**: 70, 1930.
8. THOMAS, A., and BUVAT, J. B.: Existe-t-il une encéphalite zonateuse? Herpès zoster à foyes multiples (mort au huitième jour). Paris méd **1**: 600–604, June 27, 1931.
9. CASTEL, J.: Sur un nouveau cas d'atteinte du nevraxe dans le zona. Thesis, Bosc freres, Lyon, 1935.
10. KRUMHOLZ, S., and LUHAN, J.: Encephalitis associated with herpes zoster. Arch. Neurol. & Psychiat **53**: 59–67, 1945.
11. NETTER, A.: Size observations de zonas dans l 'encéphalite léthargique. Bull. et mém. Soc. méd. d. hôp de Paris **54**: 793–798, May 19, 1930.

Systemic Effects of Oral Foci of Infection

MARTIN L. SEGAL, *Major, DC, U. S. A.*[1]
JOSEPH J. C. THOMSON, *Captain, DC, A. U. S.*[1]

FOCAL infection is defined as the condition in which a primary source of infection causes disease in some other part of the body (*1*). This is transmitted as a bacteremia or circulating allergen, which finds its way through the blood stream or lymph channels, or may spread by direct extension. Much has been written in the last four decades about the relationship between dental foci and systemic disorders. This article reports a series of patients in whom various physical symptoms and lesions were relieved by the removal of oral foci.

CASE REPORTS

Case 1.—A 28-year-old man was seen on 4 April 1949 because he could not readily adapt his vision when going from a light to a dark room. When he finally did adapt to the dark, a blind spot remained in the center of the visual field of the left eye. This condition appeared suddenly. Fundoscopic examination showed pallor in the temporal and nasal regions with blurring of the optic disk. His vision was normal under ordinary conditions, but the blind spot was unusually prominent when he tried to adapt to sunlight. The diagnoses considered were intraocular neuritis, retrobulbar neuritis, and early multiple sclerosis. The patient was referred to the dental clinic to rule out an oral focus of infection. A complete roentgenographic examination of the teeth showed a retained root tip in the upper left bicuspid region, but no noticeable area of infection surrounding it. The oral mucous membrane appeared normal; the patient had no oral subjective symptoms. Removal of the root tip resulted in a complete, uneventful recovery within 1 week. Examination 1 month later, showed no recurrence of ocular symptoms.

Case 2.—A 19-year-old man reported to the out-patient clinic on 10 December 1948 with fever, malaise, and a gastrointestinal upset of 4 days' duration. He was examined thoroughly, and an erythema was discovered over the upper dorsal and lower cervical areas of the back. This was diagnosed as a toxic erythema. The medical officer suspected a focus of infection. Roentgenograms of the teeth showed a partially erupted lower right third molar with a pericoronal

[1] General Dispensary, New York, N. Y.

866046°—50——7

abscess surrounding it. Three hundred thousand units of penicillin were administered daily until the pericronitis subsided. The tooth was removed on 15 December and the toxic erythema disappeared completely.

Case 3.—A 53-year-old man reported to the out-patient clinic on 23 March 1949 because of a patchy papulo-erythematous dermatitis of the sacral and upper inner left gluteal areas, accompanied by pruritis. There was a lichenoid plaque about 2 inches long, which was gradually becoming larger. A biopsy on 24 March showed no evidence of malignancy. On 25 April no improvement was noted and scrapings of the area were examined, but no fungi were found. Complete dental roentgenograms showed two small, extremely opaque foreign bodies with surrounding areas of radiolucency in the lower left molar area; these were about 0.1 cm. in diameter and were lodged just below the alveolar crest. When the mucoperiosteal flap was laid back and the outer layer of bone was removed, an area of granulation surrounding both foreign bodies (pieces of amalgam filling material) was found. Following their removal all skin symptoms subsided; no recurrence was noted 1 month later.

Case 4.—A 37-year-old man was seen in the out-patient clinic on 20 June 1949 because of two dry, hypertrophic, pruritic plaques, one on the left ankle, and one on the inner aspect of the left leg. The diagnoses considered were lichen planus hypertrophicus and fungus infection. The patient was treated with lotions and ointments for 1 week, with no apparent improvement. Fungus culture was negative. A roentgenogram of the teeth showed a retained root in the region of the upper right first bicuspid. Clinical examination of the mouth revealed a minute, fistulous tract in that area, from which purulent material could be expressed. The root tip was removed. Examination of the patient's leg, 1 week later, showed 75 percent healing of the two plaques. One week later the two plaques had completely disappeared.

Case 5.—A 29-year-old man reported on 20 July 1949 because of a maculopapular vesicular rash on his nose, lips, and extensor aspects of both arms, and a few scattered lesions on both lower extremities. He had had similar intermittent attacks for 6 years. Roentgenograms of the teeth disclosed a periapical lesion of the lower left cuspid. The radiogram also showed a poorly condensed inadequate root canal filling, present since 1943. The date of the filling coincided with the onset of his skin lesions. The filling was removed, the canal widened and refilled, apicoectomy accomplished, and the patient given 300,000 units of penicillin in oil. Within 10 days the skin lesions had completely disappeared.

Case 6.—A 44-year-old man was seen in the out-patient clinic on 23 June 1949 because of loss of 15 pounds in the 2 previous months, and a bilateral low back pain of 2 weeks' duration. The patient claimed that his appetite had been poor for a few weeks and that he had a general feeling of weakness with associated attacks of vertigo. Dental roentgenograms disclosed a retained root in the region of the upper left first molar. This was removed and at that time a sinus, extending from the alveolus to the left maxillary antrum, was noted. A plastic closure was performed; healing was noted 2 days later. One week later, the low back pain and attacks of vertigo had completely disappeared. There had been no recurrence.

REFERENCE

1. MILLER, S. C.: Textbook of Periodontia. 2d edition. The Blakiston Co., Philadelphia, Pa., 1943. p. 625.

About the Army Medical Department

PAUL I. ROBINSON, *Colonel, MC, U. S. A.*[1]

DURING 1949, regular members of the Medical Department have increased about as follows: Medical Corps 323 (27 percent); Dental Corps 17 (5 percent); Veterinary Corps 2 (1 percent); Medical Service Corps 14 (2 percent); Army Nurse Corps 34 (3 percent); and Women's Medical Specialist Corps 2 (1 percent). Since 1 July 1949, 293 medical officers have been brought on active duty; 65 dental officers; 11 veterinary officers; 37 medical service officers; 242 Army nurse officers; and 51 Women's medical specialists. These figures include some who transferred to the Air Force last July. They show that the regular establishment is being gradually built to strength and that there are substantial numbers in all corps who have seen fit to affiliate themselves with the Medical Department for a tour of duty. The programs which are making this progress possible deserve brief discussion.

Information concerning the Medical Department is constantly being placed before appropriate groups through the media of press releases, radio and television programs and spots, magazine articles, specially prepared pamphlets, informational letters, specific correspondence, and personal contact. All this is done in accordance with a predetermined plan. Information so disseminated is checked meticulously for accuracy and attempts to portray an honest evaluation of the subject matter covered. There is every reason to believe that this information has been received well by those most concerned. A better understanding than has been experienced for many years now exists, not only among our own personnel, but also within the various civilian medical and allied professions.

Early in the year, the American Medical Association and the Office of the Secretary of Defense, recognizing the serious shortage of medical officers facing the Army, Navy, and Air Force, launched separate pressure campaigns directed at young doctors of medicine and dentistry who had participated in Army Specialized Training and

[1] The Personnel Division, Office of the Surgeon General.

V–12 Programs during the war, but who had not served in a duty capacity with any of the Armed Forces. These campaigns resulted in a net of 158 medical and 33 dental officers who volunteered to serve for 2 years.

The internship and residency programs have lent much to the increased popularity of the Army Medical Department. These programs within the Army have been most carefully designed and fostered so that now, only 3 years after their inception, they are considered by many civilian medical educators to be equal to the best civilian programs of the nation. They are programs which will continue and become permanent parts of the military medical establishment. They are due, of course, to have considerable curtailment because the pace with which they are now training medical officers need not be continued. They need not suffer in this curtailment, however, insofar as their excellence is concerned and they, therefore, should continue to be a motivating influence in maintaining high standards of medical care within the Army.

The civilian residency program has been one of expediency. No way could be devised, 2 years ago, to train the number of specialists required within any reasonable time in the military programs. So, it was considered wise to commission doctors in civilian hospitals and allow them to continue their training there. When established, it was stated to be a 2-year program and was designed for the training of 600 officers. Actually, because of reduction in requirements for specialists, only 294 have been commissioned in the program and admissions stopped the first of 1950. (Those who are in process will be commissioned thereafter, but no other applications will be accepted.) This program, at its termination, may be regarded as completely successful. All officers in the program are members of the Regular Army Medical Corps. They all know that their resignations cannot be accepted until they have served year for year for training received. It is the sincere hope of the Surgeon General that the large majority of these officers will find the pursuit of their vocations in the Army Medical Department completely to their liking and that they will decide to remain for a career.

The civilian internship program is one of subsidization. It, too, is one of expediency. It serves partially to solve a serious problem of the Army Medical Department. This problem, simply stated, is "Who in the Medical Corps is to perform the duties of general practitioners?" The civilian intern enters the program with the understanding that he will serve 2 years in the Army after his internship. He has been placed in third priority insofar as selection for residencies are concerned. Nevertheless, the program is attractive because it offers substantial remuneration for the early years of medical experi-

ence. Eventually, this program may be determined to be most satisfactory to the individual himself because he will have had unexcelled experience in performance of physical examinations, in treatment of all types of physical and mental ailments, and in association with proficient officers in the specialties. His experience will have made him more mature and he will be in a position to predetermine accurately his career and his specialty. This program appears the only immediate source of any substantial number of young doctors to perform the general practitioner duties within the Army.

While all of this unquestioned progress was being made in professional education and medical care within the Army, other important developments have been evolving which will enter into military medicine of the future. Research and development programs are gauged to expected military medical problems. Greater knowledge regarding atomic casualties and their prevention is becoming available. Advances are being made in tables of organization to correct many of the defects which were inherent in past mobilizations. Better, more practical, and more modern equipment is being developed for field medical units. Improved management procedures are being developed for medical facilities of all types. By and large, the members of the department, as well as those in the reserve components, because of concentration on medical care activities, are not well informed of these advances. So important is military medicine that degrees in this specialty, which will compare with postgraduate university degrees, are being considered. The American Medical Association, recognizing the necessity for greater cognizance of military medicine among members of the profession, has set up for its San Francisco meeting next summer 2 half-days in its miscellaneous panel for presentation of military medical subjects. This step may be a forerunner of a specialty panel comparing to any of the others. Army, Navy, and Air Force Medical Departments will all participate in bringing this vital knowledge to the profession. And, above all, steps must be taken promptly to acquaint all regular members of the medical military establishment with these advances.

There is every indication that members of the Medical Department are responding in an intelligent manner to the preference commitments which have been made by the Congress and the Department of the Army General Staff for medical and dental officers. They are making other members of the military establishment feel that additional pay, preference in quarters assignments, and concurrent travel of their families to overseas assignments are worth while, because they are rendering medical service which has probably never been equaled in any organized section of our population. This is evidenced by a low morbidity rate and a record low hospitalization rate. It is further

evidenced by the many complimentary remarks from members of the military establishment of all ranks. It is incumbent on all of us that this fine performance continue.

Medical reserve matters have been the subject of much study in the past year. Four officers in the Army Surgeon General's office contine to spend full time on this subject. The Medical, Dental, Medical Service, and Army Nurse Corps are represented by these four officers. Early in the year the Society of Medical Consultants of World War II appointed a committee to study the problem. Dr. Frank Berry, the committee chairman, reported at the annual meeting of the Society that the difficulties might all be summed up in three words "Points—Points—Points." The Association of Military Surgeons allotted one half-day panel to the problem, in which representatives from the Staffs of the Army, Navy, and Air Force, as well as the Surgeons General of all three Services participated. Reserve officers from all parts of the nation entered into the discussion and everyone seemed to feel that the panel was definitely worth while. The Medical Advisory Committee, Office of the Secretary of Defense, has appointed a committee to further study the reserve problem. With all of the thinking entering into the reserve problem, we can expect concrete developments during the months and years ahead.

With civilian medical and allied professions, the Medical Departments of the Army, Navy, and Air Force may view with pride many coordination and unification accomplishments. The joint medical supply catalog has been in use for years. The joint purchasing office for medical items has had its fourth birthday. Medical research and development programs have been carefully coordinated for several years. Joint utilization of many Army, Navy, and Air Force medical installations is an accomplished fact. Joint staffing has been achieved in a few installations more or less on an experimental basis. Studies are in progress to create interchangeable forms and to standardize procedures. Medical representation has been established in the Office of the Secretary of Defense. Measureable economies have been effected and all of us should look forward to guidance from the new Office of Medical Services in achieving further economies in the use of personnel and other medical means.

Career planning as reported in a previous article in this series has progressed satisfactorily. Many officers have reviewed their own career plans with satisfaction. These plans are being integrated with assignments. In addition, every effort is made, not only to assure that each assignment represents a progressive step in a career, but to also consider each officer's personal situation in each assignment. Sometimes information is not sufficiently current to avoid mistakes in the latter endeavor and letters are always welcome. Insofar as trained

medical officers are concerned, the Army is particularly fortunate in having a number of large stations where teams of medical officers can work in unison to render the highest type of medical care and at the same time participate in the practical phase of military medicine, as well as preparations for mobilization.

This review of the prominent items affecting personnel activities and relations cannot help but indicate that a solid foundation has been laid for rebuilding the Army Medical Department. In this, every individual can feel that he or she has had an important part. We may now all look forward to a Medical Department which will uphold its fine traditions of the past and advance proudly with the progress of the future.

Health Discipline

Lieutenant General Sir Neil Cantlie [1] *KBE, CB, FRCS, KHP*

FIELD MARSHAL MONTGOMERY, in 1945, paid a remarkable tribute to the Royal Army Medical Corps when he wrote "to the Royal Army Medical Corps whose contribution to victory has been beyond all calculation." These are impressive words addressed by one of our greatest generals to the Army Medical Services which comprise the Royal Army Medical Corps, the Royal Army Dental Corps, and Queen Alexandra's Royal Army Nursing Corps. Never before have such glowing words been addressed by a British commander to army physicians, dentists, and nurses.

Field Marshal Montgomery stressed five main points in the contribution that the Army Medical Services had made: (*a*) Forward surgery by the field surgical units; (*b*) blood transfusions and its availability in the forward units; (*c*) skilled nursing by Army nurses in forward areas; (*d*) penicillin and sulfonamides; and (*e*) air evacuation. These are all factors that have made the difference between life and death to the wounded man and they are all concerned with curative medicine. As a further contribution to victory, a sixth point can be added which is concerned with preventive medicine. It is called health discipline and defined as measures that concern the preservation of health and the prevention of disease which are enforced by disciplinary means. These six points contributed to victory because they conserved manpower, and the recovery of sick and wounded men in total war is of great importance to total manpower. Since manpower is ultimately a deciding factor in war, an efficient medical service has an important part to play.

The effective, and, at times, overwhelming part the medical services can play in war has been gradually recognized by military commanders in the field, and this has been especially emphasized where the campaigns have been fought in countries with low standards of health. None more so than in the recent campaigns in Burma and the Pacific. The campaign in Burma in 1942 showed the extent to which military operations could be dominated by malaria. In 1943 in Burma the

[1] Director General, British Army Medical Services.

morbidity rate caused by malaria was as high as 450 cases per 1,000 per month in certain forward units in contact with the enemy, where active service conditions seriously interfered with antimalarial measures. This may seem enormous at first, but remember that it is only 15 men out of a battalion of 1,000 strong, reporting each day with malaria. Although such high rates were exceptional, rates of 200 to 250 per 1,000 per month were common among forward troops. The commanders of formations saw their forces melting away and could do nothing to prevent it.

The accepted attitude was to lay the responsibility for health on the physicians' shoulders. The prevention of malaria was a physician's duty, almost a physician's fad to which the fighting soldier paid little attention. His job was to fight and defeat the enemy. It was not appreciated that unless the battle against disease was fought and won first, there would be no troops to defeat the enemy. The battalion or regimental medical officer did his best to persuade the troops to use mosquito nets when this was possible, and to smear on repellents when they were on patrol or sentry duty. The unit sanitary personnel used Paris green and oil on pools where mosquitoes bred, and drained away stagnant water. But essentially it was a medical matter and no concern of the fighting soldier whose mind was taken up with tactical plans to defeat the enemy. It is true that the unit commander was, by regulations, responsible for the health of his troops, but he relied on his medical officer to put any measures into effect, and these were limited to what the commander thought could be adopted under the existing conditions of active service.

The campaigns in Burma and New Guinea quickly showed that combat was impossible when medical recommendations were overruled. Then came the experiments of Fairley in Australia. He proved that when one tablet of atabrine was taken daily it was impossible to get malaria. He would never report sick with malaria, no matter how many times the man was bitten by infected mosquitoes, how much he suffered exposure and fatigue, or how cold or hungry he became. When Fairley reported the success of his experiments to the American and Australian commanders in the Pacific they were quick to grasp its importance.

One commander said: "You doctors think you can prevent malaria, but you can't. I can and I'm going to." By these words, "I am going to prevent malaria," the war in the Far East was revolutionized and victory was made possible. Prevention of malaria was taken out the physician's hands and accepted by the staff. Commanders at once became responsible to assure that their soldiers did not acquire malaria, by seeing that the taking of atabrine was made a disciplinary matter. This was accomplished by requiring a daily

message from commanders of regiments, brigades, and divisions to the effect that every man had taken his atabrine. It was as simple as that, but it took a little time before the significance of the change of policy was realized. The morbidity rates for malaria dropped spectacularly. In India, which was a nonoperational area, in 2 years the malaria rate dropped from 248 to 34 per 1,000 per year. In West Africa the rate dropped from 900 in 1941 to 90 per 1,000 per year in 1945.

Commanding officers of units were responsible if the morbidity rate for malaria exceeded a maximum figure and one commander was relieved from duty for this reason. For the first time in history a unit commander was considered incompetent to command because he had allowed his men to become ineffective because of disease. Antimalarial precautions such as the taking of atabrine, the use of DDT, and the provision of mosquito nets and veils, became measures as important or more important than the supply of ammunition; and it became common practice in assault landings to land antimalarial supplies at least as soon as the ammunition, because ammunition was useless unless soldiers were there to use it, and a few hours of relaxed precaution, such as the failure of the supply of atabrine for 24 hours, would prove more dangerous than the failure to receive shells for the guns. As a result of the hard lessons of war this new doctrine, the active responsibility for the control of certain diseases rested with the general staff and no longer with the medical service evolved. This is the doctrine of health discipline.

This doctrine, and its corollary the control of disease opens up new tactical aspects of warfare. For example, if in a campaign one army has complete immunity from a particular disease because of its strict measures of health discipline, it could aim at forcing an enemy whose health measures were ineffective to fight in the most highly infected areas so that disease as well as bullets would lead to the defeat of the enemy. This occurred in Burma in 1945 when British and Indian troops were protected and the health discipline was of a high standard, while the Japanese army was decimated by malaria. We can, therefore, foresee that medical science may exercise an influence over the tactical aspects of a campaign in a country where endemic disease exists and where medical knowledge of one of the combatant forces has evolved a technique which is either unknown to or imperfectly used by the other. It follows that medical intelligence is of importance and may prove to be of great value in war. Medical intelligence means obtaining information of the diseases existing in other countries and the degree of medical knowledge that such countries possess.

The conception of the mingling of military tactics and medical science leads me to say a word about bacteriologic warfare. The bacteriologist is intimately concerned here with the actual means of destruction. We see him in the laboratory growing cultures with a view to spreading disease and death. Is this one of the prospects of advancing civilization? We as physicians have previously known no medical secrets between nations; medicine was international. There was no iron curtain until the future menace of bacteriologic warfare cast its shadow over us. Let us pray that as physicians we will never be asked to cause disease in or destroy our fellow beings. Our mission in life is to stamp out disease not to cause it, to prolong life not to shorten it.

The army medical services in war rise to a peak of importance that is sustained by the constant prospect of violent death in all its forms. The army physician becomes a savior of life and a healer of pain, while the efficiency of the medical services may be the personal concern of every soldier in the field. In the last war, as the tribute from Field Marshal Montgomery shows, our reputation has never stood so high. Our advice was accepted and enforced by commanders who wielded vast authority. It has always been the invariable experience that the lessons learned in the violence and tumult of war tend to become forgotten in the days of peace. The voice of the physician becomes a still, small voice and a generation soon grows up which lacks the personal experience of the imminence of sudden mutilation or death.

It must be one of our great tasks in peace to keep alive this flame of health discipline, which was first kindled in the embers of war. We must not allow that great contribution to victory in the field to be cast aside and forgotten. Human nature, being forgetful of the past, will have to be constantly reminded of the part we have to play. Our soldiers in peace do not die of malaria, or scrub typhus, or dysentery—diseases which may decimate armies in the field. We can only accomplish this task by constantly emphasizing the lessons of the past to the present and future generations. We must keep our officers aware of the problems of health discipline and we must do this by lectures and by health education, which should be part of the syllabus of all combatant officers' training. I hope this policy will bring home to our staff officers who are responsible for future planning, that the medical services must be called in at an early stage and not forgotten, which has happened in the past. Our medical intelligence must be fully developed and the importance of this branch of military medicine should be reemphasized. We must employ it with caution to avoid divulging secret information. For example, when, in 1942, the invasion of a certain country was being planned in great se-

crecy, the director of medical services of the force was engaged in a survey of the malarial problems that might be encountered. Wishing to know the habits of all the endemic varieties of anopheline mosquitoes so that effective control measures could be planned, he asked the entomologist what he knew of the habits of Anopheles "X." The entomologist replied, "What on earth do you want to know that for? It only exists in a limited area in . . ." (mentioning the country where the invasion was to occur). The director of medical services had to draw in his horns pretty quickly to avoid a serious leak in the invasion plan.

Health discipline must cover all those diseases that can be controlled by regulations enforced by authority. So far malaria has been dealt with exclusively, but there are other diseases that the advance of medical science can add to this category. It would be rash to make predictions, but it is suggested that scrub typhus, which was met in Burma, with its 20 percent mortality may shortly be controlled in the same way. Chloromycetin used prophylatically has been found to give favorable results. The active search for new antibiotics will no doubt produce new drugs which will unlock those doors which are still shut. If a drug effective as a prophylactic against bacillary dysentery could be produced then a standard of health discipline could be enforced that would do away with one of the scourges of armies in the past.

As for venereal disease, is it too much to hope that a drug may be discovered which when given prophylactically will prevent at least one of the venereal diseases? Will the time come when the disease controlled by a drug in this way would be made a matter of health discipline and would pass from medical to disciplinary control? This is dangerous ground, because moral principles are involved.

One of the medical consultants in Burma, from his personal experience in the last war, laid the strongest emphasis on the necessity to shift our natural bias from curative to preventive medicine. He maintained that by multiplying the sanitary personnel in the forward areas and by intensive propaganda and health education we could attain a degree of health discipline that would materially reduce our hospital beds and hospital staffs. This is a point of view that merits serious thought and it is believed that greater efforts to inculcate health education followed by effective measures of health discipline would be sound. Recently the title of the Director of Hygiene at the War Office has been changed to Director of Army Health. It is believed that this alteration in title represents the change that is taking place in the conception of the duties of the Director, for emphasis is now laid on the study of positive health or what has sometimes been called functional medicine in distinction to curative and preventive medicine.

Finally, let us consider the measures of preventive medicine and health discipline that the soldier of future wars will be expected to undergo. He will be immunized against smallpox, the enteric group of fevers, tetanus, diphtheria, yellow fever, louse-borne typhus, and if necessary, cholera and plague. Tuberculosis may soon be added to this list. From the point of view of health discipline he will be protected against malaria and probably scrub typhus, and possibly at least one of the venereal diseases. Let us not forget the chemotherapeutic agents that each man may swallow if wounded.

We need not feel dissatisfied with the prospects of protection against disease afforded to our soldiers, but how much more satisfactory if a prophylactic drug effective against the dysenteries could be added to this list. Surely with the advances we can expect in antibiotics we cannot discard this as impossible. In conclusion, there is a story of an eminent American thinker who was once asked how he would have made the world different if he had been God. He replied, "To begin with I should have made health infectious instead of disease."

In the absence of perfection such as this, one of the great tasks of military medicine must be to send the soldier to war protected from those diseases that can be prevented, and by the powers of health discipline effectively wielded by the supreme commander in the field. The importance of health discipline has been stressed because we cannot allow the lessons of the last war to be forgotten. When for the first time in history a combatant officer was considered unfit to command a unit on the grounds that he had allowed his men to become ineffective through disease, a new day in military medicine dawned. The clouds of forgetfulness must not be allowed to overshadow the brightness of that day.

The Surgeon in the Army: A Career Pattern

WARNER F. BOWERS, *Colonel MC, U.S.A.*[1]

CURRENT emphasis on board certification has created a situation in which many desirable assignments are unwanted because they cannot be applied to board credit. Is it possible that everything we do must be checked off for "credit"? It would appear that what was originally meant as a stepping stone has become accepted as an end in itself. Most informed observers agree that there is too much emphasis on specialization too soon, but how the tide is to be turned is not apparent. A Board of Military Medicine might be the answer to the needs of the service for men who are specialists in the various aspects of preventive and curative medicine and might also satisfy the general yearning for some sort of certification. Such certification might include training in various fields to produce physicians with a well-balanced knowledge of clinical medicine, but here we come up against the general practitioner type of training which has been so unpopular that no one volunteers for the course.

This article is written because many of the problems referred to the Office of the Surgeon General indicate a lack of appreciation by the inquirer of what he desires and why. Each surgeon or would-be-surgeon must determine for himself what his aim in life is and then set out to attain it. If such a goal is superficially conceived, continual shifts in emphasis and direction become necessary, and life progresses from one crisis to another. On the other hand, if a career is planned in a mature manner and each phase of training and experience is used constructively, the maximum amount of personal serenity and satisfaction will result. The following discussion is intended to be based on common sense and a realistic approach to the problem.

REQUIREMENTS FOR SPECIALISTS

There are several methods by which the number of specialists required in the Army has been calculated, but each method arrives at

[1] Surgical Consultants Division, Office of the Surgeon General, U. S. Army.

practically the same figure. In spite of this, and because of the great overemphasis on specialty board certification, there is continual pressure to increase the length of training, the volume of the program, and the number of residents in each hospital. Is it better to train only the required number of specialists and make specialty training unavailable to many who desire it, or to train all who want it and not have assignments available when their training is completed? With the current trend toward specialty training, few men are entering the service for any other reason than to pursue a residency course. In the United States, about 22 percent of the practicing physicians are specialists. The original figure set for the Army was 30 percent. The fact that, as time goes on, more and more of the men in command and staff positions will be volunteers from the professional fields is taken into consideration. This is already evident; a number of hospital commanders are certified specialists in various clinical fields. If, as it seems, we will not be able to get 70 percent of the Corps to be general practitioners, a plan must be devised whereby we can accomplish our mission with a higher percentage of specialists. What would happen if all medical officers became certified specialists? Everyone is fully aware of the terrific cry that went up during and after the war, that doctors were not used in their specialty and consequently there were allegations of great waste of trained men. Can we again voluntarily embark on a program that will inevitably end in such criticism? Further, what of the wartime mission of the Medical Corps? Can we justify training all our officers as bedside specialists to the neglect of field medicine, which is presumably our special sphere of interest? What would happen in wartime if we had only clinical specialists in the Medical Corps? There can be little question that civilian physicians can provide patient care in the service, but who will argue that a clinical practitioner with no service experience can do as well as a Regular Army officer in staff and command positions?

Recently, our position has been reevaluated in the following light. If we cannot get general duty medical officers, what is the answer? One answer seems to be to train sufficient men in surgical fields so that all or nearly all of the assignees on surgical services will be trained men. As chief of service, would you rather have general duty medical officers or trained men as ward officers? The answer is obvious. Strangely enough, when this type of computation was made, only a slight increase in the number of trained surgeons required was noted. A checking of current rosters of all hospitals, putting certified specialists in all possible positions, again gave an almost identical figure. A third study based on experience in visiting most of these installations and assigning specialists according to the known volume of work in each field, again gave a closely comparable figure.

A knotty problem involves the use of D-grade specialists. It seems self-evident that our military men and their dependents are entitled to the best possible medical care. In certain specialties it seems that a little knowledge may not be harmful, but is this true in surgery? Is it safe to say that men with 9 months' training can be entrusted to open the peritoneal cavity without competent supervision? The answer, that it has been done with no serious results for many years, means that such a course may be followed of necessity, but not necessarily as a matter of choice. Certainly anyone who performs major operations should be prepared to cope with any situation that may arise. Consequently, it is sound to believe that D specialists should be only assistant residents in training. What if an officer fails to make suitable progress in the residency program and is dropped from training after a year? Should he be sent out with a D rating? This has been done in the past with serious results. Such a man, on reporting to a station hospital for assignment, may be the only one present for duty with that MOS and consequently he becomes chief of service. If the man has been relieved from training for incompetence or unfitness for the work, can we justify his assignment as chief of service in that field?

TYPES OF SURGICAL TRAINING

There are several types of surgical training, but surgery in the Army will not come of age and will not be recognized in the national professional societies until we have chiefs of service who are research-minded teachers, steeped in the everyday application of the basic sciences to everyday clinical practice. The "pick-up" or preceptorship type of training does not produce such men, but does produce surgical technicians who know an operation for each disease. Unfortunately, it is impossible to appreciate something that has not been personally experienced and consequently, "pick-up" surgeons do not even recognize their inadequacies. Unusual insight was demonstrated recently by a surgeon who failed to pass the board examinations in surgery when he said that men of his caliber simply cannot think in terms of the pathologic physiology of surgical diseases, as is required today.

The 3- or 4-year residency, based on adequate, supervised experience, with increasing responsibility and founded on the basic science approach with grounding in research, is the best type of surgical training that we know today. Unfortunately, many who are responsible for administering such a program have not had the benefit of such training themselves and as a result, a certain amount of cross-purpose results. Many hospitals look on residents as a cheap answer to the personnel shortage and confuse this with the fact that duty

personnel are needed for the care of patients. It is unfair to sacrifice approved training in this way and some other solution needs to be found. The fact that residents in urology, otolaryngology, neurosurgery, and certain other specialties need a year of general surgery does not mean that they can be assigned to out-patient clinics and other places just because someone has to do the work there. It frequently happens that an additional period of training must be given because of such assignments.

A similar point of misunderstanding arises over the assignment of senior residents. The boards stress the element of increasing responsibility throughout the period of training but the assignment of senior residents in surgery as ward officers in orthopedics, gynecology, or otolaryngology is not acceptable because they have not had this rotation or because residents have not applied for training in that field. If hospitals have so many senior residents in surgery that they can be farmed out on other services, it is planned to reduce the assigned senior residents next year by that number. It is appreciated that ward officers are needed in specialties where few apply for training, but senior residents in surgery are not the answer. In certain hospitals, senior residents are getting too little experience because the chief of service or the consultants do too much. Why a chief of service in a teaching hospital does simple herniotomies is difficult to understand. Here again, continuance of this practice will result in a decrease in the number of residents assigned to such a service.

A similar problem relates to the use of gynecologic patients to provide operative experience for residents in general surgery. Recently, several residents in obstetrics and gynecology have had to be continued in training for abnormally long periods because of such misuse of clinical material. If clinical material in general surgery is meager, the number of residents should be reduced. It is a generally accepted policy that gynecology and obstetrics go together in the training program. If general surgeons can be trained in gynecology without jeopardizing the other residency program, there is no valid objection. In some instances, space allocations have made it necessary to transfer residents to two or more hospitals during their training period and in such instances the receiving hospital almost invariably requests that such trainees be put back to a lower level of training. It seems unlikely that all men trained by some other chief of service will be inferior. Some training in other hospitals must be considered acceptable.

The place of clinical research in a training program is important but widely misunderstood. All residents should be encouraged and required to engage in research in clinical problems. Case reports with review of the literature, statistical analyses of groups of cases, and

866046°—50——8

discussion of experiences with new apparatus or drugs and similar subjects are suitable. It is not anticipated that fundamental discoveries will be made by all surgeons and it should be realized that the resident himself benefits most from clinical research. He gains clarity of thought, ability to think a problem through, practice in presenting his material, and experience in medical writing. This is sufficient reward.

If a D rating is granted for minimum training, a C rating for completion of the residency training, and a B rating for success in board certification, how then does one attain an A? Certainly not by occupying a certain assignment for 1, 2, or even 3 years. An A man is one who has done something to advance the specialty by making a real contribution in the field of research, an outstanding teacher, or someone nationally recognized in professional groups. Repetition of mistakes over a period of years does not constitute experience nor does passage of time as a B necessarily add up to an A.

ACCREDITED PRACTICE TIME AND THE EVACUATION POLICY

From time to time we should ask ourselves the purpose for which we are training men in surgical fields. A common-sense answer would seem to be that we want trained men to go out and practice their specialty in the various Army medical installations. This answer, however, leads through many ramifications to conclusions that affect the entire residency program. For over a year we have been working on the problem of obtaining sanction from the various specialty boards for accrediting the practice time required before the final examination is taken. This phase has been practically completed and most of the boards are willing to grant credit after proper residency training for experience in the field of special training in any Army hospital of sufficient size to guarantee an adequate volume of cases and if supervision by civilian consultants or assigned board-certified chiefs is possible. This means then that at any Army hospital where a certified chief of service or a trained man requiring practice credit is assigned, the evacuation policy must permit enough major work so that the practice time will be recognized as adequate by the board and so that the chief of service, if board certified, will do sufficient work in his chosen field to enable him to feel that the hard years of training, study, and examinations have been worth the effort.

As more and more men complete training and are assigned to the smaller hospitals, more major work will be done there and fewer cases will be transferred to the general hospitals. This is desirable from the standpoint of the personnel and their dependents at the post level and, furthermore, the effect on morale throughout the Medical Corps when we no longer have to apologize to line organizations for lack of

specialists of our own will be beneficial. This will also allow us to assume more of the teaching and consultant load and will enable us to lean less heavily on the civilian consultant program. This restoration of professional confidence in our own men will increase the dignity and stature of our Corps, which has suffered greatly since the personnel exodus after the war.

A natural outcome of this inevitable trend will be a cut-back in the volume of the residency training program, a discontinuance of some of the programs in some of the hospitals and possibly the closing of some of the smaller general hospitals. There are some who are unwilling to face this development, but, obviously, training depends on case load and if that drops, approved training decreases in proportion. Those who want to have their cake and eat it too fall into three main groups: Those who personally profit in rank or ego from an empire will resist to the last ditch; those who are interested in training for training's sake cannot face the prospects of a decrease in the program; finally, there are those who feel that a training program produces better patient care and must be maintained for that reason. This attitude is justified but has no bearing on the problem since a training program of a high type can be maintained without residents who are working for credit. For example, during the war at every station and general hospital where the author was chief of service, he insisted on a university hospital type of training program in which everyone participated. Such a program may evoke more actual interest than a program in which residents are marking off attendance for credit. Even if residency training should decrease, this should not be allowed to have any effect on the continuance of a program of conferences, rounds, and other teaching exercises.

SURGICAL DUTY ASSIGNMENTS

Some interesting problems arise in attempting to assign men on completion of residency training. Although we have affirmed our belief that we are training men so that they can go out into the various medical installations and practice their specialties, we find almost without exception that these men expect a general hospital assignment, preferably as chief of service, and accept a station hospital assignment with poor grace. What can be the reason for this except a degree of mental immaturity and failure to think the problem through clearly? Obviously, chiefs of service in the general hospitals will not be replaced by each new crop of residents as they emerge from training. Also, since all personnel allotment slots in the teaching hospitals except for the chiefs, are filled with residents, under the present system there is no vacancy for the assignment of duty personnel.

A variation of this problem lies in the expectation of each senior resident that he will continue with the same volume and caliber of cases that he has been handling as a senior resident. This assumption also is fallacious. Let us first consider the philosophy of residency training. It is not anticipated that a resident necessarily will perform every procedure known to his field during training, but that he will obtain sufficient experience and a sufficiently broad background so that he can intelligently manage whatever problems may arise later. It is not anticipated that a resident will operate at maximum capacity from the moment of completion of residency training. Let us compare these matters with the usual sequence in civil life. How many civilian residents have stepped from training into a professorship of surgery or even position as a chief of staff in a smaller hospital? How then can it be expected that the assignment situation in the service would be different? How many civilian residents have performed splenectomies, gastrectomies, combined abdomino-perineal excisions, and pneumonectomies in their first few years of private or university practice after completion of training? How then can it be expected that such a volume of cases will be available to the service physician just out of training? If we have so much trouble getting sufficient cases for the senior resident, how can he but realize that he will do less in his first years of assignment? Proper perspective is difficult to attain, but we must strive to see ourselves in proper relation to the total situation and without undue magnification of our own image or an unrealistic feeling that we will greatly outstrip our contemporaries in knowledge or skill. Unfortunately, most of us are distressingly similar in capabilities.

It seems obvious, therefore, that most men will receive station hospital assignments on completion of training and that on completion of the required practice time they will become certified specialists. Again, perspective is lost and the board certificate is assumed to be the culmination of a career when as a matter of fact it is simply a key that may unlock the door to greater opportunity. Let us hold no brief for board certification. All it means is that a man has passed through certain prescribed training in an approved institution and has made a suitable impression on a group of examiners who see the candidate under abnormal conditions and for a very short time. The certificate, then, is simply a clearance as it were, indicating that the man is ready for better things. No certificate bestows experience, judgment, or mental maturity, which are so profoundly important in service chiefs. It must be obvious then, that the newly certified specialist will become chief of a service when and if he proves himself worthy of such an assignment and when there is a vacancy in such a position. Such an assignment, then, will depend entirely on worth

and ability instead of seniority and incumbency, which is as it should be. In other words, resting on the laurels should be reserved for the period of retirement—the best possible care for the patient must always be the paramount consideration. What of the surgeon who fails of certification? Obviously, he cannot hope to become chief of a service in a teaching hospital but may become chief in a nonteaching hospital or may become post surgeon and chief of surgery in a smaller hospital. Here again, such an officer will remain in the practice of surgery or its specialties only as long as he is the best qualified officer available for the position.

As residency training decreases with the production of trained men, duty assignments will become available in general hospitals again and the best qualified men will have an opportunity to work up to assignment as chief through demonstrated ability. A pertinent word of self-caution is "Let's not get too good too soon."

GENERAL OUTLOOK

Having struggled to become a specialist, what is there in prospect? Data seem to indicate that the average medical officer, assuming a 30-year career, may expect to spend 10 years in assignments not requiring specialized training. He will spend another 4 years in assignments requiring only limited specialized skill, such as ward officer in a general hospital or a resident in training. He may then expect to spend about 12 years in assignments requiring advanced specialized training. About half of this time would be spent in assignments requiring board certification. The average medical officer might expect to spend 4 of his 30 years in a command or staff position. It must be emphasized that these figures pertain only to the average medical officer and within this average there will be doctors who either because they do not desire or cannot qualify, will never receive specialized training and will spend their entire career as general practitioners, while a few may perform major operations for 30 years. How does this compare with civilian practice? Much overemphasis has been placed on the fact that medical officers may be required to perform duties not directly related to patient-care without realizing that large numbers of civilian physicians are engaged in other than professional work. Figures supplied by the Resources Analysis Division, Office of the Surgeon General, show that as of 1 August 1947 4,400 physicians in the United States or 2 percent of the total, were employed by insurance companies, industrial firms, or in other administrative positions. Another 2,654 or 1 percent are superintendents of hospitals. Of the total of 197,605 physicians in the United States, only 74 percent are in private practice and of these only 22 percent are limiting their practice to a specialty. How then can it be said that administration is peculiar

to the Army physician or that our original estimate that about 30 percent of the Corps should be specialists is so far wrong?

A young physician is never capable of seeing himself as an older man. Most young surgeons assume that they will operate till the day they are forced to retire, but a realistic approach shows that while some surgeons operate to the end of their career, many become deans of schools, officials of national groups and specialty boards, hospital superintendents, and so forth. If this is the majority trend, why is it not logical to assume that most of us will satisfy our inclination for surgery before 30 years have passed? If it takes us less than 30 years to come to a realization that there is more to surgery than operating, what avenues are open beside administrative, staff, or command positions, which are so abhorrent to some?

Teaching is a field in which one can grow old gracefully, with dignity and personal satisfaction. The pleasure of seeing one's students develop in knowledge and skill is a real compensation for many other disappointments. Research is another field from which much stimulation and satisfaction can be derived. An older man with a broad background is in an enviable position to direct research and make contributions of lasting value. Consultant work is a newer field, quite closely allied to teaching and of proved value. The great success of the consultant program in the last war and its continuation in the succeeding years makes it unlikely that this field will decrease in scope. An important maxim that is conducive to a happy and purposeful life is "Know where you want to go and be on your way."

THE PHYSICIAN MUST KNOW WHAT HIS PREDECESSORS HAVE KNOWN IF HE DOES NOT WISH TO DECEIVE BOTH HIMSELF AND OTHERS.—*Hippocrates.*

EDITORIAL

The (Ab)use of Morphine

The time-honored concept that morphine is the essence of treatment in the severely wounded should be critically examined.

It has been traditional to assume that the severely wounded man inevitably has severe pain and that pain is in direct proportion to the extent of the injury. The careful observations of Beecher have not borne out this premise. His factual material based on the observation of 215 recently wounded men in the combat zone emphasizes the inaccuracy of the general belief that all severely wounded men have severe pain. In the light of his observations the premise that morphine must be given routinely to all injured persons appears to be faulty.

Beecher observed 215 wounded soldiers; 50 had compound fractures of long bones; 50 had extensive soft tissue wounds; 50 had penetrating wounds of the thorax; 50 had penetrating wounds of the abdomen; and 15 had wounds of the cerebrum. The time since wounding varied from 11.3 to 12.5 hours in those with compound fractures and soft tissue wounds, and from 7.2 to 9.8 hours in the others. The average total dose of morphine given varied from 25 mg. for those with thoracic wounds to 29 mg. for those with abdominal wounds; in cerebral wounds a total dose of 19.8 mg. had been administered. The latest dose of morphine varied from an average of 19.5 mg. in those with soft tissue wounds to 25 mg. in those with penetrating wounds of the abdomen. The time since the latest dose of morphine varied from 4.8 hours in the abdominal wounds to 7.2 hours in those with soft tissue wounds.

The over-all effects of injury showed that:

	69 patients, or	32.1%, had no pain
	55 patients, or	25.6%, had slight pain
	40 patients, or	18.6%, had moderate pain
	51 patients, or	23.7%, had bad pain
Total	215	100.0%

A close parallelism existed in the number wanting pain relief and those reporting bad pain:

Bad pain reported:	
Yes	51 cases, or 23.7%
No	164 cases, or 76.3%
Pain relief therapy wanted:	
Yes	58 cases, or 27.0%
No	157 cases, or 73.0%

The following percentages of patients in the first group said that they had bad pain:

Penetrating cerebral wounds	7%
Penetrating wounds of the thorax	12%
Extensive soft-tissue injury	16%
Compound fractures of long bone	24%
Penetrating abdominal wounds	48%

Of the 51 patients who reported severe pain, 5 had had no morphine and the other 46 has an average of 24.5 mg. for the most recent dose.

None of the 215 men had evidence of shock at the time of questioning. Only 25 percent said their pain was severe enough to require pain relief. The remaining 75 percent did not need pain relief despite the fact that the time since the last dose varied from 4.8 hours in penetrating abdominal wounds to 7.2 hours in extensive soft tissue wounds.

The data presented strongly suggest that morphine is all too often used indiscriminately in the belief that severe wounds are inevitably associated with intense pain and further, it appears that morphine is probably often used for the relief of symptoms other than pain.

The prevailing use of morphine in shock is also based upon an erroneous concept. Morphine is contra-indicated in shock. In shock (traumatic or hemorrhagic), arterial vasoconstriction, low circulating blood volume, poor cardiac output, slow capillary blood flow, reduced peripheral circulation, and depression of blood pressure occurs. Morphine, even in moderate doses, in shock, further depresses blood pressure and so lowers an already low circulating blood volume, and further diminishes cardiac output; it antagonizes vasoconstriction (a compensating process in shock), and dilates cutaneous vessels, and acts to further slow the blood flow and increase capillary stagnation. Morphine depresses the medullary center and stimulates the reflexes and the spinal functions. Morphine very often produces nausea, vomiting, and fainting, particularly in those with surgical lesions; it slows the pulse by vagus stimulation; it diminishes oxygen consumption and carbon dioxide output, and lessens the response to CO_2 and to asphyxia—all these actions aggravate existing shock.

Severely wounded men unquestionably have some degree of shock although no symptoms of shock may be apparent. (It must be recalled that symptoms such as pallor, sweating, nausea, restlessness, or delirium are manifestations of late severe shock.)

The severely wounded, because of shock, are not prone to complain of pain but instead are restless and apprehensive and show excitement—the symptoms of circulatory insufficiency and cerebral hypoxia. Further, repeated doses of morphine given to patients in shock subcutaneously or intramuscularly remain in the tissues and are not absorbed until peripheral circulatory efficiency is restored. Then the accumulated doses of morphine are simultaneously absorbed and morphine poisoning occurs.

Zweifach and Hershey have shown that one of the major determinants of the character of shock is the type of anesthetic agent employed. In dogs in shock the intravenous injection of morphine produced a gradual slowing of the capillary flow and a slow depression of blood pressure which could be alleviated only with blood transfusions.

Although morphine is unquestionably of value in severe pain, no more than 10 to 15 mg. should be given. The optimum effect is obtained with 10 mg.; little more relief is obtained with doses greater than 10 or 15 mg. It must be given intravenously if immediate action is desired.

The restlessness, excitement, and apprehension of the severely wounded in shock is due to cerebral hypoxia and only blood transfusions will relieve this. Barbiturates or paraldehyde and not morphine should be used to relieve excitement and apprehension in the severely wounded.

It is well known that hypnosis or suggestion can alter the perception to pain and that the effects of an analgesic drug can be modified by psychological factors. Reassurance of the severely wounded patient, the physician's or attendant's attitude being one of conviction and that he knows what he is about, plus the use of a placebo when required will modify the patient's reaction to pain and allay his fears. Such measures in conjunction with the use of supportive measures will be effective in the treatment of pain and shock.

BIBLIOGRAPHY

1. BEECHER, H. K.: Pain in men wounded in battle. Ann. Surg. 123: 96–105, Jan. 1946.
2. ZWEIFACH, B. W., and HERSHEY, S. G.: Predisposing action of anesthetic agents on vascular responses in hemorrhagic shock. Surg. Gynec. & Obst. 88: 469, Oct. 1949.

3. SOLLMAN, T. H.: A Manual of Pharmacology and Its Applications to Therapeutics and Toxicology. 7th edition. W. B. Saunders Co., Philadelphia, Pa., 1948.

4. BEECHER, H. K.: Resuscitation and anesthesia. Anesthesiology 7: 644–650, Nov. 1946.

BOOK REVIEWS AND BOOKS RECEIVED

Publishers submitting books for review are requested to address them as follows:

The Editor,
UNITED STATES ARMED FORCES MEDICAL JOURNAL,
Bureau of Medicine and Surgery, Navy Department,
Washington 25, D. C.
(For review)

ATOMIC MEDICINE, edited by Charles F. Behrens, M. D., *Captain, MC, U. S. Navy, Director, Atomic Defense Division, Bureau of Medicine and Surgery, Navy Department; Medical Officer in Command, Naval Medical Research Institute, National Medical Center, Bethesda, Md.* 416 pages; illustrated. Thomas Nelson & Sons, New York, N. Y., publishers, 1949. Price $7.50.

This volume is a compilation of several chapters by various authors, all of whom are more or less engaged in research and are considered experts in their respective fields: radiology; radiophysics; radiochemistry; biochemistry; dentistry; and pathology. The subject is assembled in an orderly sequence and contains a vast fund of information, so condensed and so written, that, with but few exceptions, it can be readily understood by the average physician for whom this book is primarily intended. There is some unnecessary repetition, but this can be readily corrected in future editions. This book deals with the fundamental principles of nuclear physics, the atom and its components, the means of artificially altering its structure, thereby its behavior, the fundamental concepts underlying the inception and conception of the atom bomb, the effects produced by its explosions at Hiroshima and Nagasaki, with special emphasis upon the pathological effects on human beings, and suggestive methods of coping with such an atomic disaster, should one occur in the future. The application of atomic energy in the form of radioactive isotopes in research and therapeutics is adequately covered. Each chapter is concluded with a long bibliography making reference easy for the reader who desires additional information. In reading this book, one cannot help but come to the conclusion that some of the "sting" and the mystery once surrounding the atomic bomb has been dissipated. This unique book is recommended reading for every physician. —*Commander M. W. Mason (MC) U. S. N.*

HEMATOLOGY, by Cyrus C. Sturgis, M. D., *Professor of Internal Medicine, Chairman of the Department of Internal Medicine, University of Michigan Medical School, and Director of the Thomas Henry Simpson Memorial Institute for Medical Research, University of Michigan.* 915 pages, illustrated. Charles C Thomas, Springfield, Ill., publisher, 1948. Price $12.50.

This book covers comprehensively the accumulated knowledge of hematology from the earliest beginnings of scientific medicine down to the latest developments in the subject. An attempt has been made to discuss the disease states

on the basis of the broad aspect of clinical practice rather than from the more narrow viewpoint of a highly specialized branch of internal medicine.

Great strides have been made in this field of medicine in the past few decades. Therefore, it is essential that a carefully compiled bibliography such as is presented in this textbook be available for the student of hematology or for the practitioner.—*Lieutenant Commander K. P. Knudtson (MC) U. S. N.*

HISTOPATHOLOGY OF IRRADIATION FROM EXTERNAL AND INTERNAL SOURCES. Edited by William Bloom, M. D., *Professor of Anatomy, Department of Anatomy and Institute of Radiobiology and Biophysics, University of Chicago.* 808 pages, illustrated. McGraw-Hill Book Co., Inc., New York, N. Y., publishers, 1948. Price $8.

In reality the best review of this book is that by the editor, himself, in which he gives a very impartial and honest appraisal of the type of work that was done. the aims, and the accomplishments. To elaborate somewhat more, this book is an original recording of extensive studies done in a field of great interest today. It will be an invaluable aid to workers in the field of radiobiology in general. The book's greatest value is the excellent bibliography of the existing literature up to 1945. The extensive amount of work that was done by the Chicago group and presented in this book will act as the "jumping-off" spot for future work in various other institutions and laboratories. The illustrations are plentiful and well chosen and, in general, are well reproduced by the publisher. The section on blood and blood-forming organs is particularly well illustrated by excellent color drawings. These illustrations describe adequately the morphologic changes that take place in the hematopoietic organs. The experimental work is clearly outlined and the results are tabulated in such a way that it is easy to refer to them and understand the data.—*Lieutenant Commander E. P. Cronkite (MC) U. S. N.*

PHYSICAL MEDICINE IN GENERAL PRACTICE, by William Bierman, M. D., *Attending Physical Therapist, Mount Sinai Hospital; Assistant Clinical Professor of Medicine, Columbia University.* With a chapter on Medical Rehabilitation by Dr. Sidney Licht. 2d edition, revised and enlarged. 686 pages, 310 illustrations. Paul B. Hoeber, Inc., New York, N. Y., publishers, 1947. Price $8.

This is an especially useful book for the physician not too familiar with the means and methods of physical medicine. Each agent used in physical medicine is discussed under a separate heading. The physiological rationale for each type of agent used is well discussed. The subjects of physical medicine are covered in clear and concise language. Much of the basic principles and descriptive physics are omitted.

The subject of medical rehabilitation is well covered in a chapter by Sidney Licht. The care of paraplegic and amputee patients is adequately explained. There is also an excellent chapter on occupational therapy.

The final nine chapters of the book deal specifically with physical medicine as applied to diseases of the locomotor, nervous, cardiovascular, digestive, genito-urinary, and respiratory systems, and of the ear, nose, throat, and skin.

—*Lt. G. E. Vaupel (MC) U. S. N.*

OCCUPATIONAL MEDICINE AND INDUSTRIAL HYGIENE, by Rutherford T. Johnstone, A. B., M. D.; *Consultant in Industrial Health; Lecturer at the University of California, Los Angeles; formerly Assistant Professor of Medicine, University of Pittsburgh School of Medicine; formerly Director of Department of Occupational Diseases, Golden State Hospital.* 604 pages, with 117 illustrations, 7 in color. The C. V. Mosby Co., St. Louis, Mo., publishers, 1948. Price $10.

This new work on industrial medicine is designed to combine the clinical aspects of the occupational diseases with a description of the industrial processes, their appraisal and control. Its material is unusually well selected for use by the average naval medical officer confronted with industrial hazards and occupational diseases.

Part 1 deals with the general principles of industrial medicine, medicolegal aspects, and the accurate establishment of diagnoses of occupational disease, to avoid the "conjectural approach." A useful table of laboratory procedures indicated for various industrial poisons concludes this short section.

The bulk of the book discusses, in turn, the numerous specific disease-producing substances, in sections on gaseous and vaporous agents, the metals, and the dusts, followed by a discussion of special disease and industrial problems of unusual prevalence or importance, the dermatoses, synthetic plastics, special processes new to many medical officers, and the principles of industrial hygiene and protective devices including appraisal of hazards.

In addition to full discussion of such new problems as DDT, 2 4–D, Beryllium, BAL, and recent plastics, the book commends itself to the average reader because of free use of excellent illustrative case histories, analyses of hazardous situations, and simple instructions for treatment. A short but pertinent bibliography concludes each chapter. The appendix, which gives the chemical composition of numerous products bearing nonrevealing trade names, a table of maximum permissible concentrations of 86 toxic agents, and lower explosive limits of 71 inflammable vapors will be a convenient source of valuable information.

The book is recommended as a basic volume for the library of any naval activity having industrial health problems.—*Capt. R. W. Babione (MC) U. S. N.*

STUDIES IN AIR HYGIENE, by R. B. Bourdillon, C. B. E., M. C., A. F. C., D. M.; O. M. Lidwell, D. Phil.; and J. E. Lovelock, Ph. D., with W. C. Cawston, L. Colebrook, M. B., F. R. C. O. G., F. R. S.; Surgeon Commander F. P. Ellis, O. B. E., M. D., R. N.; M. Van Den Ende, M. B., Ph. D.; R. E. Glover, M. Sc., F. R. C. V. S.; A. M. MacFarlan, M. B., B. Ch.; A. A. Miles, F. R. C. P.; W. F. Raymond, B. A.; E. Schuster, O. B. E., D. Sc.; and J. C. Thomas, M. R. C. S., L. R. C. P. 356 pages, illustrated. His Majesty's Stationery Office, London, Eng., publishers, 1948. Price $1.30.

The authors give (*a*) very detailed descriptions of laboratory and field apparatus for air sampling to determine the bacterial contamination of air, (*b*) practical methods of air disinfection (chemicals, ultraviolet radiation, heat, and filtration), (*c*) some results of field studies to measure levels of bacterial contamination of air in operating rooms, homes, factories, and ships, and (*d*) the results of a few animal experiments on transmission of airborne infections.

Surgeons will be interested in the data presented to show the necessity for creating a slight positive pressure in operating rooms to prevent contamination from the air sucked in under doors, etc. The relative merits and testing methods of various surgical masks are also discussed. The devices and experimental designs presented here can be utilized by surgeons who might like to make studies of wound infections related to contamination of the air in the operating rooms. The testing devices record time so that high concentrations of bacteria can be related to various maneuvers or circumstances. The internist will be interested in such topics as the method of estimating tubercle bacilli in air.

This report is so well written that the reader can easily understand the particular section of interest to him without constant reference to other sections.

—*Capt. H. G. Shepler (MC) U. S. N.*

MALIGNANT DISEASE AND ITS TREATMENT BY RADIUM, by Sir Stanford Cade, K. B. E., C. B., F. R. C. S., M. R. C. P., *Surgeon, Westminister Hospital, Mount Vernon Hospital and Radium Institute; Lecturer in Surgery, Westminister Hospital Medical School and formerly Examiner in Surgery, University of London; Member of the Court of Examiners, late Hunterian Professor and Arris and Gale Lecturer, Royal College of Surgeons of England; Member of the National Radium Commission and Trust; Consultant in Surgery to the Royal Air Force;* with a foreword by Sir Ernest Rock Carling, F. R. C. P., F. R. C. S., F. F. R., *Consulting Surgeon and Vice President, Westminister Hospital.* Vol. I, 2d edition. 383 pages, illustrated. The Williams & Wilkins Co., Baltimore, Md., publishers, 1948. Price $12.50.

The first edition of this work was published in 1940 and was included in a single volume, while the second and present edition, has been divided into four separate books. Volume I, with which this report is concerned, deals with radium and its effects. It is divided into two parts: (1) The Radioactivity of Radium, which deals with the physical principles of radiation, and (2) The Biological Effects of Radiation, derived from observation of the effects of radiation, and clinical and experimental work utilizing tissue cultures.

The opening discussion of Part I is devoted to general considerations of cancer and radium; it is short and to the point, contains numerous pertinent statements relative to the indications for radium therapy, comparison of results between surgery and radium therapy in certain types of lesions, as well as the use of both forms of therapy in a given case. This discussion represents the opinion of one who is both a surgeon and a radium therapist.

Next is a short chapter devoted to the composition of radium followed by a more lengthy discussion on Technique of Radium Therapy which deals with types of radium application excluding radium teletherapy, and contains some isodose curves.

Under the chapter on Radium Dosimetry, the various methods for measuring the ionization current are considered, including the various types of apparatus. There is also a discussion of surface, cavitary, and interstitial methods of radium application. The method of measuring the dosage rate from radium plaques and radium teletherapy are both discussed in separate chapters.

Part II begins with a discussion on radiosensitivity involving the various physical factors that affect the radiosensitivity of tissues, then a short chapter on the mode of action of radiation, based on observations from experimental, clinical, and histological evidence. The effects of radium emanation on normal tissues is discussed in the section which follows, in which the effects on various types of tissue are discussed. Next, is a general discussion of the effect of radium on malignant tumors, as determined by microscopic examination of serial biopsies, and a classification of tumors derived from their relative radiosensitivity. It amply illustrates the importance of doing serial biopsies during the course of treatment.

A new chapter entitled "Tissue Culture and Experimental Radiology" written by F. G. Spear, M. A., M. D., D. M. R. E., has been added at this point. It deals with observations on the effects of radiation upon tissues cultivated "in vitro." Comparison of the effects of continuous irradiation with that of spaced doses on cultures under varying conditions is given in this chapter, together with similar observations on tissues "in vivo."

The final chapter deals with "The Dangers of Radium and Protection," as applied to the patient and to the radium worker. To better illustrate the importance of observing proper safeguards, 20 case histories are given.

In general, the book appears to be well written by one who is amply qualified to write on all phases of the subject. Not only has the author drawn his conclusions from a wide personal experience, but he also seems to have a rather broad acquaintance with the literature on this subject. It is a good book on which to lay the foundation for a working knowledge of the application of radium, and is written in a clear and concise style. The print is good size for reading and the paper and binding are of good quality.—*Commander C. D. Burroughs (MC) U. S. N.*

BOOKS RECEIVED

Receipt of the following books is acknowledged. As far as practicable, these will be reviewed at a later date.

DISEASES OF THE NERVOUS SYSTEM, by W. Russell Brain, D. M. (Oxon.), F. R. C. P. (London), *Physician to the London Hospital and to the Maida Vale Hospital for Nervous Diseases; Sometime Neurologist to the Infants' Hospital and Physician to the Royal London Ophthalmic Hospital; Theodore Williams Scholar in Physiology in the University of Oxford; Price Scholar in Anatomy and Physiology at the London Hospital.* 3d edition. 987 pages, illustrated. Oxford University Press, New York, N. Y., publishers, 1947. Price $10.75.

LIVING ANATOMY, A Photographic Atlas of Muscles in Action and Surface Contours, by R. D. Lockhart, M. D., Ch. M., *Regius Professor of Anatomy, University of Aberdeen; Formerly Professor of Anatomy, University of Birmingham; Examiner in Anatomy to the Universities of Aberdeen, Birmingham, Glasgow, Manchester, St. Andrews, and Sheffield; the Conjoint Board of the Royal Colleges of Physicians and Surgeons, and the Chartered Society of Physiotherapy.* 71 pages, illustrated. Oxford University Press, New York, N. Y., publishers, 1948. Price $4.

ADOLESCENCE PROBLEMS, A Handbook for Physicians, Parents, and Teachers, by William S. Sadler, M. D., F. A. P. A., Chicago. *Consulting Psychiatrist, Columbus Hospital; Fellow of the American Psychiatric Association, The American Medical Association, The American Association for the Advancement of Science; Member of the American Psychopathological Association.* 466 pages. The C. V. Mosby Co., St. Louis, Mo., publishers, 1948. Price $4.75.

MANUAL OF THE INTERNATIONAL STATISTICAL CLASSIFICATION OF DISEASES, INJURIES, AND CAUSES OF DEATH. Sixth Revision of the International Lists of Diseases and Causes of Death. Adopted 1948. Vol. I. World Health Organization, Geneva, Switzerland, publishers, 1948.

FUNDAMENTALS OF BODY MECHANICS AND CONDITIONING, An Illustrated Teaching Manual, by Mabel Lee, B. S., LL. D., *Professor of Physical Education and Director of Physical Education for Women, University of Nebraska,* and Miriam M. Wagner, B. A., M. A., *Assistant Professor and Director of Health and Physical Education, University of Kansas City.* 377 pages with 70 figures. W. B. Saunders Co., Philadelphia, Pa., publishers, 1949. Price $4.50.

NEOPLASMS OF BONE AND RELATED CONDITIONS, Their Etiology, Pathogenesis, Diagnosis, and Treatment, by Bradley L. Coley, M. D., *Attending Surgeon, Bone Tumor Department, Memorial Hospital for Cancer and Allied Diseases, Assistant Professor of Clinical Surgery, Cornell University.* 622 illustrations and 53 tables. Paul B. Hoeber, Inc., New York, N. Y., publishers, 1949. Price $17.50.

THE EPIDEMIOLOGY OF HEMOLYTIC STREPTOCOCCUS During World War II in the United States Navy, by Alvin F. Coburn, M. D., *The Rheumatic Fever Research Institute, Northwestern University Medical School,* and Donald C. Young, M. D., *Medical Director, Communicable Disease Service, Herman Kiefer Hospital.* 220 pages. The Williams & Wilkins Co., Baltimore, Md., publishers, 1949. Price $4.

BRITISH SURGICAL PRACTICE, Vol. 5, under the general editorship of Sir Ernest Rock Carling, F. R. C. S., F. R. C. P., *Consulting Surgeon, Westminster Hospital,* and J. Paterson Ross, M. S., F. R. C. S., *Surgeon and Director of Surgical Clinical Unit, St. Bartholomew's Hospital, Professor of Surgery, University of London.* In 8 volumes. The C. V. Mosby Co., St. Louis, Mo., publishers, 1948. Price $15.

MEDICAL ETYMOLOGY, The History and Derivation of Medical Terms for Students of Medicine, Dentistry, and Nursing, by O. H. Perry Pepper, M. D., *Professor of Medicine, University of Pennsylvania.* 263 pages. W. B. Saunders Co., Philadelphia, Pa., publishers, 1949. Price $5.50.

CURRENT THERAPY 1949, Latest Approved Methods of Treatment for the Practicing Physician. Howard F. Conn, M. D., Editor; Consulting Editors: M. Edward Davis; Vincent J. Derbes; Garfield G. Duncan; Hugh J. Jewett; William J. Kerr; Perrin H. Long; H. Houston Merritt; Paul A. O'Leary; Walter L. Palmer; Hobart A. Reimann; Cyrus C. Sturgis; and Robert H. Williams. 672 pages. W. B. Saunders Co., Philadelphia, Pa , publisher, 1949. Price -10.

THE EPITOME OF ANDREAS VESALIUS, Translated from the Latin with Preface and Introduction by L. R. Lind, Ph. D., *University of Kansas;* with Anatomical Notes by C. W. Asling, M. D., Ph. D., *University of California;* and a Foreword by the late Logan Clendening, M. D. 103 pages of translation and 28 pages of Latin text. The Macmillan Co., New York, N. Y., publishers, 1949. Price $7.50.

BLOOD TRANSFUSION, edited by Geoffrey Keynes; 8 contributors. 574 pages; illustrated. The Williams & Wilkins Co., Baltimore, Md., publishers, 1949. Price $12.50.

PUBLIC HEALTH IN THE WORLD TODAY, edited by James Stevens Simons, *Brigadier General, U. S. Army, Retired, Dean, Harvard School of Public Health;* Assistant Editor, Irene M. Kensey; with Foreword by James Bryant Conant, *President of Harvard University.* 22 contributors. 332 pages, illustrated. Harvard University Press, Cambridge, Mass., publishers, 1949. Price $5.

INDUSTRIAL TOXICOLOGY, by Alice Hamilton, M. D., *Assistant Professor Emeritus of Industrial Medicine, Harvard School of Public Health, Boston, Mass.,* and Harriet L. Hardy, M. D., *Physician to the Division of Occupational Hygiene, Massachusetts Department of Labor and Industries; Assistant in Medicine, Massachusetts General Hospital, Boston; Consultant, Los Alamos Scientific Laboratory, University of California, Los Alamos, N. Mex.; Instructor, Department of Industrial Hygiene, Harvard School of Public Health, Boston, Mass.* 2d edition, revised and enlarged. 574 pages. Paul B. Hoeber, Inc., New York, N. Y., publishers, 1949. Price $7.50.

THE ADRENAL GLAND, by Frank A. Hartman, Ph. D., *Research Professor of Physiology, the Ohio State University;* and Katharine A. Brownell, Ph. D., *Instructor in Physiology, The Ohio State University, Columbus, Ohio.* 581 pages, 72 illustrations. Lea & Febiger, Philadelphia, Pa., publishers, 1949. Price $12.

THE KINESIOLOGY OF CORRECTIVE EXERCISE, by Gertrude Hawley, M. A., *formerly Head of Corrective Gymnastics at Stanford and Northwestern Universities.* 2d edition, thoroughly revised. 192 pages with 107 illustrations. Lea & Febiger, Philadelphia, Pa., publishers, 1949. Price $3.75.

MANUAL OF CLINICAL LABORATORY METHODS, by Opal Hepler, Ph. D., M. D., *Associate Professor of Pathology, Northwestern University Medical School; Director of the Clinical Laboratories of the Montgomery Ward Clinics and Passavant Memorial Hospital; Consultant in Clinical Pathology at Children's Memorial Hospital, Chicago, Ill.,* with a foreword by James P. Simmonds, Ph. D., M. D. 4th edition. 387 pages, illustrated. Charles C Thomas, Springfield, Ill., publishers, 1949. Price $8.50.

THE PREMATURE INFANT, Medical and Nursing Care, by Julius H. Hess, M. D., *Professor Emeritus, Department of Pediatrics, University of Illinois College of Medicine; Senior Attending Pediatrician, Michael Reese Hospital, and Consulting Pediatrician, Cook County Hospital;* and Evelyn C. Lundeen, R. N., *Supervisor, Premature Infant Station, Sarah Morris Hospital Station of Michael Reese Hospital, Chicago.* 2d edition. 381 pages, illustrated. J. B. Lippincott Company, Philadelphia, Pa., publishers, 1949. Price $6.

A TEXTBOOK OF ORAL PATHOLOGY, by Thomas J. Hill, D. D. S., *Professor of Clinical Oral Pathology and Therapeutics, Western Reserve University, Cleveland, Ohio.* 4th edition, thoroughly revised. 398 pages, with 314 engravings. Lea & Febiger, Philadelphia, Pa., publishers, 1949. Price $7.50.

REVIEW OF DENTISTRY, Questions and Answers. Edited by James T. Ginn, B. S., D. D. S., *Professor and Chief of Division of Operative Dentistry, Professor of Oral Medicine, University of Tennessee College of Dentistry, Memphis; Formerly Assistant Professor of Pathology, Histology, and Radiology, Loyola University of the South, School of Dentistry, New Orleans; Formerly a Carnegie Fellow in Dentistry, University of Rochester School of Medicine and Dentistry, Rochester, N. Y.* 810 pages. The C. V. Mosby Co., St. Louis, Mo., publishers, 1949. Price $5.75.

○